ACUTE INJURIES OF THE HEAD

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THEIR DIAGNOSIS, TREATMENT COMPLICATIONS AND SEQUELS

BY

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TO

PROFESSOR GEOFFREY JEFFERSON
TO WHOM BRITISH NEURO-
SURGERY AND MANY YOUNG
NEURO-SURGEONS OWE SO MUCH

PREFACE

THIS book has been written primarily for those who are responsible for the treatment of acute cerebral trauma and who have not received a special training in neurosurgery or in neurology. It will also be of value, I believe, to senior students about to be confronted with the complexities of injuries of the head in the near future

An effort has been made to present a continuous picture of the various problems concerned, from the moment of the infliction of violence to the stage of complete recovery or of invalidism. This is important, since what happens in the acute phases materially affects what follows, both as regards complications and sequels.

In the chapter on diagnosis the temptation to oversimplify what is essentially a complex problem has been avoided, since anything but a true presentation of the facts only leads to confusion.

It has, of course, in a book of this size been impossible to cover every variety of fracture of the skull and cerebral injury, but I hope a starting-point has been made from which a rational approach to the subject is possible. The references are not exhaustive; they were not meant to be so. They do, however, give an introduction to the literature on each of the subjects considered and will lead to most of the important ones not mentioned.

Most of the observations on which this book is based were made at the Stockport Infirmary, where the fullest facilities have always been granted me for clinical and research work. Many of the autopsies were done in conjunction with my friend Mr Andrew McGill, and by the courtesy of Mr Ferns, the Coroner.

To Dr F. M. R Walshe I am sincerely grateful for reading the typescript and for the many valuable criticisms he made. I felt that if I could satisfy him I could await future judgments with confidence.

I would also like to thank Mr Norman M. Dott for much useful advice and encouragement in the early stages

The drawings have all been made by Miss D. Davison, and to her I am most grateful, not only for her skill which speaks for itself but also for her patience.

To Mr Macmillan many writers in recent times must be thankful for the high standards he has set in publications, and I appreciate also his sustained understanding.

Without Miss Bousfield's and Miss Hudleston's help this book would never have been completed

G. F. ROWBOTHAM.

FAIRHOLME,
WYLAM
1942

CONTENTS

FOREWORD

THE medical profession has been increasingly aware of the need of an authentic treatise on head injuries; and it will assuredly welcome this volume as meeting that need admirably With modern development of mechanisation in transport and industry, accidental injuries have progressively and alarmingly increased. During the past three decades orthopædic surgery has become increasingly organised, and in the last few years its application to the casualties of industry and transport has been promoted and developed on a national scale. Thus an important advance has been achieved and is being developed in caring for the injured limbs of the community.

What of the injured heads? Head injury has a high incidence in modern civilian casualties. Its toll in fatalities and—still worse—in serious and permanent mental and physical incapacity is heavy. That this devastation can be very significantly mitigated by the combined application of neurology, psychiatry, and neurosurgery to these casualties is recognised. Yet no adequate treatise on the subject had appeared in Britain until Mr Rowbotham met the challenge with this volume. Still less had any widely organised effort been made to meet the special needs of the head-injured.

In this state of unpreparedness in this particular field, the present war has descended upon us. Whereas in peace time the proportion of disability due to brain diseases is relatively high as compared with physical injuries of the brain, in war—and especially in modern "total war"—the proportion due to physical violence may be expected to be relatively much higher. Thus while a clamant need for a book such as this existed before the war, it is now an acute need. Britain to-day owes much to the author for meeting it. His book is a work of art, in which a sympathetic care and understanding of the injured is the strongest note, yet now it is also as a weapon in our hands by means of which a not inconsiderable part of our enemies' activities will be frustrated. Meantime the advent of war has jolted the "powers that be" into organising nationally for the care of the head-injured, and "Head Injury Centres" have been established and are developing under the guidance of such men as our author. With this book

to indicate the means and this organisation to put them into effect we shall better meet the stress of war; and we shall also emerge upon the peace better equipped to care for our head-injured than we were in 1939.

Why had the care of our head-injured lagged behind other medical activities? The devious ways of advance of medical science are indeed curious. The specialists obviously equipped to deal with these casualties had failed to take them in hand. Neurologists and psychiatrists saw little of them, presumably because they were labelled "surgical" from the circumstance that they had acquired their disabilities by physical violence. Neurosurgeons—a small and recently established sect—were for the most part fully occupied with the surgery of brain disease, and most of them had not yet extended their activities to deal with brain injuries. Thus, in general, the head-injured did not receive attention from those specially equipped to deal with their needs, and the care of head injuries remained a Cinderella of medical science.

The author dedicates this volume to his teacher, Professor Geoffrey Jefferson. He does so with good cause. Among the many wise and beneficient activities of this outstanding figure of British medicine, the care of the head-injured has been a constant preoccupation Jefferson did good work on head injuries and made important contributions to this subject in the last war, and has continued this in his school at Manchester since. His senior pupil, our author, has not failed to realise the importance of the subject, and has so ordered his affairs that he has acquired a very large experience of it.

This extensive personal experience of all aspects of the care of head injuries, coupled with a notable capacity for clear observation, will be found to mark every page of the volume

The author is known as a neurological surgeon His book shows a wide grasp of the problems raised by brain injury—neurological, psychiatric, and social, as well as surgical He makes it clear that he who would treat these patients successfully must have considerable training in and knowledge of each of these sciences, and must work in close collaboration with other specialists in them. I believe that this penetrating exposition of the scope of the subject will in the future prove of even greater import than the excellent practical instruction which this book supplies and which we so urgently require at the present time.

NORMAN M DOTT.

EDINBURGH,
1942

CHAPTER I

THE MECHANISMS OF INJURIES OF THE HEAD

THE physics governing cranial and cerebral injuries are complicated and this can be readily understood, since the head is a complex body and since the violences to which it is subjected are variable in magnitude, direction and area of application. In actual practice, sufficient data to permit a precise reconstruction of the accident are rarely forthcoming, but none the less a visualisation, particularly of the ways in which injurious forces may be transmitted to the brain, is essential if the problems of cerebral trauma are to be approached logically. Fortunately the mechanisms concerned in fracture of the skull and brain damage have aroused a good deal of interest, with the result that much has been written on this subject.[1-7]

FRACTURES OF THE SKULL

Since bone possesses the property of elasticity, it will bend whenever a force of sufficient magnitude is applied to it under the right conditions, but whether it breaks depends on the degree of bending to which it is subjected Bending certainly is the means by which most fractures of the skull are produced, and the injuring force acts either by deforming a circumscribed area of bone or by distorting the whole skull. The exact manner in which the bone breaks is determined by the fact that its tensile strength is less than its power to resist compression. Therefore whichever table of the skull happens to be on the convexity of a bend, and thus subjected to stretch, will be the one to

[1] Von Bergmann, E "System of Practical Surgery," 1
[2] Duret, H "Traumatismes craniocérébraux" *Librairie Felix Alcan*, **2**, Part I Paris, 1920
[3] Saucerotte, N "Mémoire sur les contrecoups dans les lésions de la tête" *Mém pour le Prix de l'Acad roy Chir*, *Paris*, 1778, **10**, 282
[4] Chopart, F "Memoire sur les contrecoups dans les lesions de la tête" *Mém pour le Prix de l'Acad roy Chir*, *Paris*, 1778, **11**, 137
[5] Felizet, G M "Recherches anatomiques et experimentales sur les fractures du crâne" Paris, 1873
[6] Polis, A *Rev Chir*, 1894, **14**, 273, 645
[7] Miller, G G "Cerebral Concussion" *Arch Surg Chicago*, 1927, **14**, 891.

fracture first. This sequence of events in the solution of osseous continuity is well illustrated in the snapping of a stick across the knee.

However, before the skull can be locally deformed or generally distorted, and thereby fractured, it must offer some kind of resistance to the violence which is applied to it; otherwise the head would merely be moved through space without undergoing structural change. In other words, a counter force must act on the skull at the same time as the injuring force if a fracture is to occur When the head is crushed between two external objects, the points of action and reaction, as the sites of application of the injuring and reactionary forces may be called, are obvious Crushing in this way, however, happens to be a very rare type of accident. In the majority of cases the point of reaction must be in the region of the craniospinal junction, as this is the only point at which the head is tethered and resistance possible. This leads us to a consideration of the mode of attachment of the head to the body The occipito-atlantal and the atlanto-axial articulations are so constructed that the movements of the head in relation to the spinal column have the same freedom as a ball and socket joint. Therefore, within a limited range, the head can move away from an injuring force and tend to escape damage, but not fully, since a certain amount of force is always expended when a body is put into motion from a position of rest or when its motion is increased

Movements beyond the range of the occipito-atlanto-axial joints are prevented by the strong relatively inelastic occipito-atlanto-axial ligaments, and these structures also resist vertically or longitudinally directed forces which tend to lift the skull from, or drive it across, the spinal column. Thus the tension of the craniospinal ligaments sometimes acts as a counter to the injuring force.

In ordinary circumstances of accident, forces tending to knock the head off the spinal column are rare, whereas forces directed downwards and tending to crush the head against the spinal column are extremely common. Thus the rigid support which the skull receives at its occipital condyles is one of the most important anatomical features concerned in the mechanisms of fracture of the skull and injury to the brain

Fractures due to Local Deformation.—The many factors concerned in local deformations of the skull are most obvious when definite indentations occur. A small mass striking the head squarely will, if travelling at a sufficiently great speed, drive inwards a piece of bone shaped as a cone-like indentation (Fig. 1). At the apex of such a cone the inner table will be stretched,

whereas the outer table will be compressed, and thus it is the inner table which fractures first Fracture of the outer table follows as the force continues to act, and the completed fracture line or lines must run from the central point radially. Occasionally fractures restricted to the inner table, unless revealed by radiography, pass unrecognised at the time of the injury, although later they may give rise to traumatic epilepsy if a spicule



Fig 1

A typical indentation due to local violence The inner table is fractured at the apex and the outer table at the periphery

of bone has happened to pierce the dura Sparing of the outer table in these cases is explained by the mechanics of the fracture rather than by any particular brittleness of the inner table itself.

At the periphery of the indentation the bone is bent in the opposite direction, the convexity of the bend being outwards, and here the outer table fractures first; also the fracture lines produced tend to run circularly to enclose the base of the indentation. When the injuring force has not been expended after it has produced a complete circular fracture, a piece of bone fragmented by the radial fracture lines will be loosened and then

depressed to form a typical comminuted depressed fracture (Fig. 2). Massive depressed fractures are caused by large objects travelling at great speed and they are usually overwhelming and fatal.

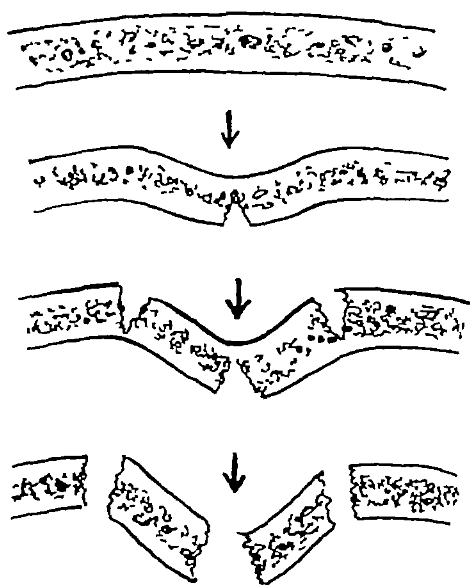


FIG 2
The sequence of events in fracture by local deformity

Fractures due to General Deformation.—In virtue of its shape and the physical properties of bone the skull behaves in some degree like an elastic sphere. Therefore whenever it is compressed, for example laterally, there is a shortening in the line of pressure, while the vertical and longitudinal diameters are increased, which means that parts of the skull distant from the site of application of the injuring force are bulged and may fracture by bending.

The head, as shown above, may be compressed in one of two ways:—

(*a*) Between two external objects, such as the ground and the wheel of a motor car.
(*b*) Between an external object and the spinal column.

The latter method of compression is the more important, as it is the commoner one, and is best illustrated in motor-car accidents when an occupant of the car is thrown from his seat. In some phase of the accident the head strikes against a resistance, possibly the roof of the car, the windscreen or the ground and comes to rest, whereas the body, in virtue of its momentum, continues to travel onwards, with the result that the weight of the body through the spinal column is thrust against the occipital condyles (Fig. 3). The same mode of compression occurs when the body is at rest and a heavy object, such as masonry, falls on to the top of the head, driving the skull downwards on to the condyles of the atlas, a type of accident which commonly happens to civilians during air raids when the building above them is blown down

Fractures due to local deformation are commonly associated with those due to general distortion For example, in falls on the head, apart from fracture by bursting, a circular fracture around the occipital condyles, due to local deformation, may detach the basi-occiput and allow it to be driven into the intracranial cavity

These points were very clearly illustrated in one of my cases when a motor cyclist, travelling at speed, came into collision

with a motor car and was thrown through the air a distance of fifteen yards before his head struck the ground. The impact was so great that his skull was almost completely flattened and brain tissue oozed from wide longitudinal fissures in the vault, which were the result of bursting. Furthermore, the basi-occiput



FIG 3

Crushing between the spine and an external object is the means by which fracture of the skull is usually produced In this mechanism local, as well as general, distortion occurs

loosened by fracture due to local bending had been driven inwards and was resting against the dome of the calvarium

FRACTURE PATTERNS

As v. Bruns[1] has pointed out, if the skull were equally thick and equally elastic and formed a true sphere, the lines of fracture could be calculated mathematically, and these would depend on the magnitude and direction of the force and on the size of the

[1] von Bruns, P "Die Chirurgischen Krankheiten und Verletzungen des Gehirns und seiner Umhullungen" *Handbuch der praktischen Chirurgie fur Arzte und Wundärzte* Tübingen, 1854, 1

body inflicting the violence. Although the physics governing such a hypothetical system are helpful in visualising what happens in injuries of the head, important modifications have to be made for anatomical irregularities. In actual fact, the skull is not a true sphere, neither is it a homogeneous body offering a uniform resistance, but is composed of relatively thin panels of bone enclosed within strong buttresses, and this explains to some extent how fracture patterns are so numerous and why at first sight they appear subject to no rule.



FIG 4
The weak panels and strong buttresses of the vault

The Influence of Strengthening Buttresses.—In the vault there are vertical thickenings at the glabella, external angular processes, mastoid bones and external occipital protuberance, and these are united by six arches, three on each side, viz , the supra-orbital ridge in front, the curved lines of the occiput behind and the temporal crests at the side Also there is a stout anteroposterior arch of bone at the top of the skull in the middle line protecting the sagittal sinus The sheets of bone in the base are much thinner than those in the vault, but, on the other hand, they are enclosed within extremely strong buttresses One buttress runs anteroposteriorly in the middle line broken by the foramen magnum and sphenoidal air sinus The petrous bones run inwards

and forwards from the sides, their weak point being externally where they enclose the middle ear. More anteriorly, the thickened edges of the sphenoid wings run inwards and backwards, the weak point here lying internally where the two wings separate to enclose the sphenoidal fissure. The general disposition of the various bands of thickening and areas of relative thinness are shown in the accompanying drawings which were made after transillumination of the skull (Figs. 4 and 5).

In view of the above anatomical facts it can be readily understood how fracture lines often take a zigzag course, for even though a force tends to travel in the direction in which it was initiated, it also tends to follow the lines of least resistance, and will split up into components to avoid traversing a stout barrier of bone.

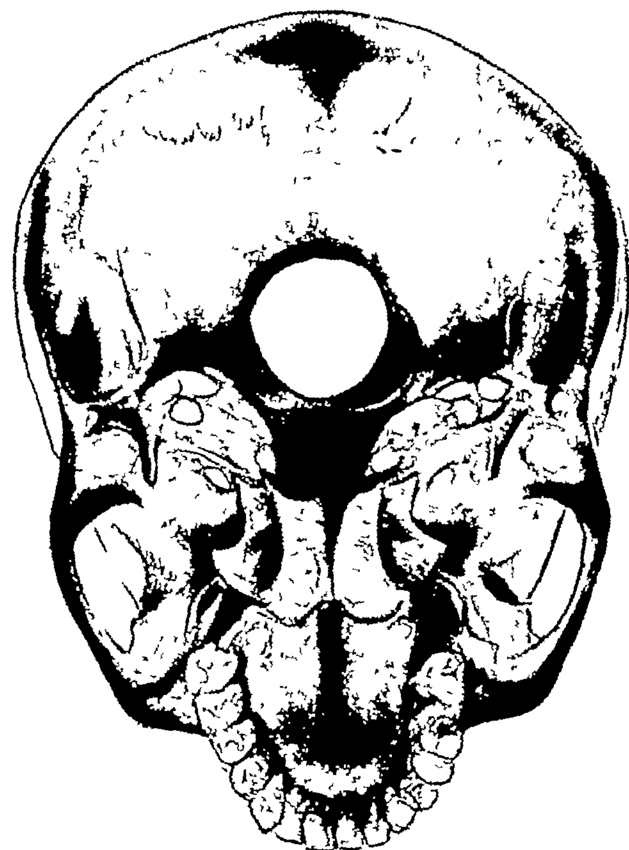


FIG 5
The weak panels and strong buttresses of the base

Fracture patterns in the base are strongly influenced by the petrous buttress, since fracture lines which approach it from the middle or posterior fossa are turned either towards its apex or base, according to the angle at which they strike it Only when the forces are overwhelmingly great is the middle of the body of the petrous bone ruptured, and forces of such magnitude usually cause fatal injuries Many basal fractures converge on and overrun the pituitary fossa, and this is not surprising, for not only is it central in position and, therefore, necessarily often in the line of injuring forces, but it is also the point to which fracture lines will be deflected by the numerous buttresses which radiate from it

The base of the skull, unlike the vault, is weakened by numerous foramina, and fracture lines commonly open into these various

openings. The sphenoidal fissure is most frequently affected, although the foramen magnum is by no means spared in spite of its thickened margins Occasionally the whole floor of the middle fossa is loosened by a fracture which runs inwards along the anterior margin of the petrous bone to the foramen ovale, then forwards on the side of the pituitary fossa and finally outwards through the sphenoidal fissure, to end in the region of the pterion.



FIG 6

The type of fracture produced by forces directed towards the vertex The arrows indicate the direction of the injuring force It is of interest to note that the fracture lines run parallel with the base Also they are widely opened and presumably act as natural decompressions, particularly as loose bony fragments are lifted and not depressed towards the brain

The Influence of the Site of Application and Direction of the Injuring Force.—A force applied to the vault and directed towards the vertex tends to lift off a dome of bone in the same way as a knife lifts off the top of an egg, and thus extensive fractures of the calvarium may be produced which run parallel to the base This type of longitudinal fracture occurs in falls in which the head strikes a projecting object in the phase when the feet are dependent, and since the upper segment of bone is lifted away

from the brain and not driven towards the base of the skull, cerebral injuries tend to be minimal (Figs. 6 and 7)

Slicing injuries, although not very common, are important since they are liable to produce compound fractures The classical sabre cut is being replaced by slashing wounds inflicted by aeroplane propellers, and one case has recently been described in the literature in which an instructor had part of his frontal bone and

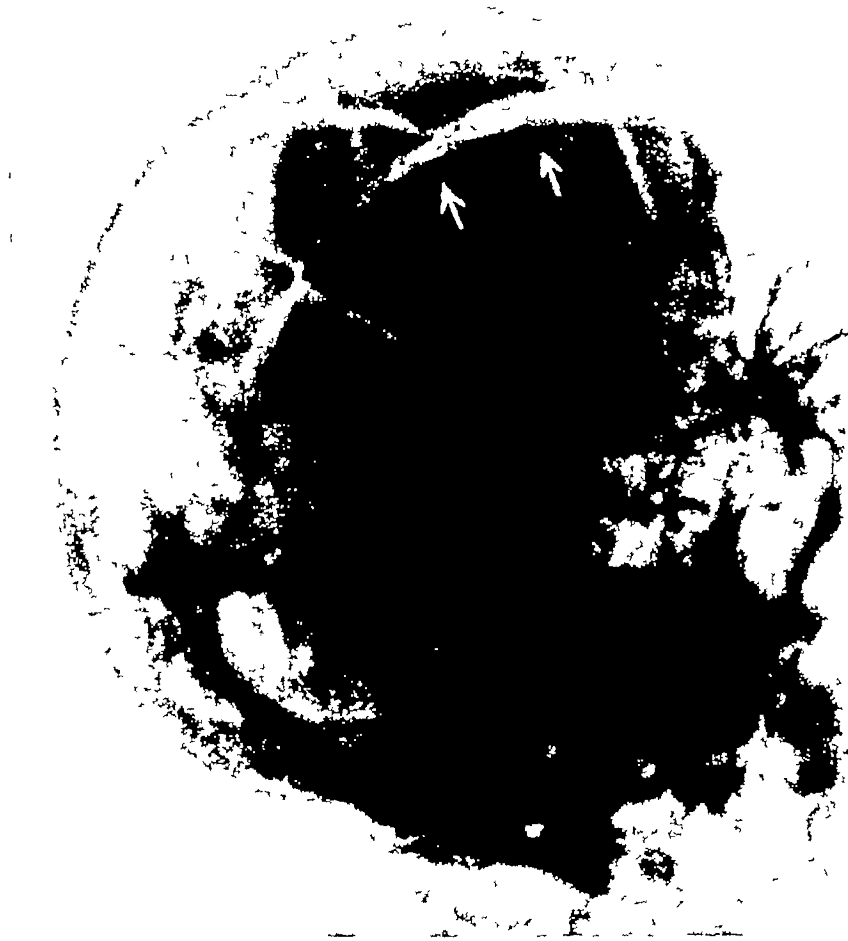


FIG 7

An injuring force striking the skull vertically will first of all put the occipito-atlantal ligaments on the stretch Then, if the force is not expended, the dome of the calvarium, lying above the site of impact, will tend to be lifted from the base Fractures produced in this way are circular in the transverse plane Often they are best demonstrated by an oblique X-ray view as shown above

lobe removed by such an injury, he did not lose consciousness immediately and ultimately made a satisfactory recovery.[1]

In one of my own cases a youth received a similar injury when attempting to change the tyre of a motor lorry The metal band clamping the tyre in position flew loose as he levered it away from the rim of the wheel and struck him in the orbit beneath

[1] D'Arcy, T N. "A Case of Severe Head Injury" *Jour. R Nav Med Serv*, 1936, **22**, 242

the supra-orbital ridge and sliced off the front of his head (Fig. 8).

Blows on the chin occasionally fracture the glenoid fossa, but it is rare for the mandibular condyle to be driven into the intracranial cavity, partly because the head rides the blow, and chiefly because closure of the jaws transfers the force to the face and prevents the mandible from moving upwards. The serious effects of blows on the superior maxilla are becoming more widely



Fig 8
Loss of bone due to a slicing injury

recognised, for although the central part of the bone enclosing the antrum of Highmore crushes easily and absorbs even a severe shock, its internal angular process is strong and readily transmits forces to the non-resistant cribriform plate which is easily fractured. Occasionally the cribriform plate is loosened and tilted inwards, leading to rupture of the olfactory filaments with loss of all but trigeminal taste (salt, sour, bitter, sweet), and often to laceration of the dura, with the danger of meningitis, since the fracture opens into the nose

An oblique blow of great severity applied to one side of the back of the head will start a fracture in the underlying posterior fossa which crosses the middle line to enter the middle fossa of

the opposite side, possibly to end in the anterior fossa. Messerer,[1] in particular, has stressed the influence of the direction of the injuring force on the line of fracture and has enunciated a law to the effect that longitudinally or transversely directed forces always produce fractures in the corresponding axis.

Rawlings [2] was of the opinion that most basal fractures are the result of forces applied at the level of the base initiating a chisel action which prises open the bone in the line of the injuring force. Some certainly are produced in this way, but it is by no means the only possible mechanism, although it is an important one. Aran [3] showed that many fractures of the vault are vertical and that they are continuous with fractures in the nearest basal fossa. On this evidence the theory of irradiation was evolved, though not by Aran, which postulates that basal fractures are purely the lower continuations of fractures which originate in the vault

In fact, basal fractures may be produced in a variety of ways :—

(*a*) By forces applied directly at the level of the base.
(*b*) By general distortion of the skull wherever the forces are applied.
(*c*) By extension from the vault
(*d*) By forces applied to the base through the spinal column or face

There may be no external bruising, and often details of the mode of the accident are lacking. Occasionally evidence from the above sources may help in deciding by which mechanism the fracture was produced, or an opinion may be formed from the pattern of the fracture itself. For example, a ring fracture around the basi-occiput is due to the thrust of the spinal column. In severe injuries the complexity of the fracture pattern often suggests that several factors have been active and have combined to produce the extensive and comminuted fractures of the base sometimes found (Fig. 9).

Apart from anatomical irregularities and the variability of the site of application and direction of the injuring force, there are probably other factors, though these are difficult to prove, which explain how fracture lines occasionally run at all angles or stop and then start again. In road accidents in particular the head may be struck more than once under different conditions, the final fracture pattern being the result of more than one injury

[1] Messerer "Ueber Elasticität und Festigkeit der Menschlichen Knocken" Stuttgart, 1880
[2] Rawlings, L B "Surgery of the Skull and Brain" London, 1912
[3] Aran, F A "Recherches sur la fracture de la base du crâne" *Arch Gén Méd*, 1844, **6**, 180, 309

Moreover the skull is often struck over a wide area, which means that the injuring force is applied simultaneously at many points that may change in position during the different phases of the injury as the skull flattens and deforms.

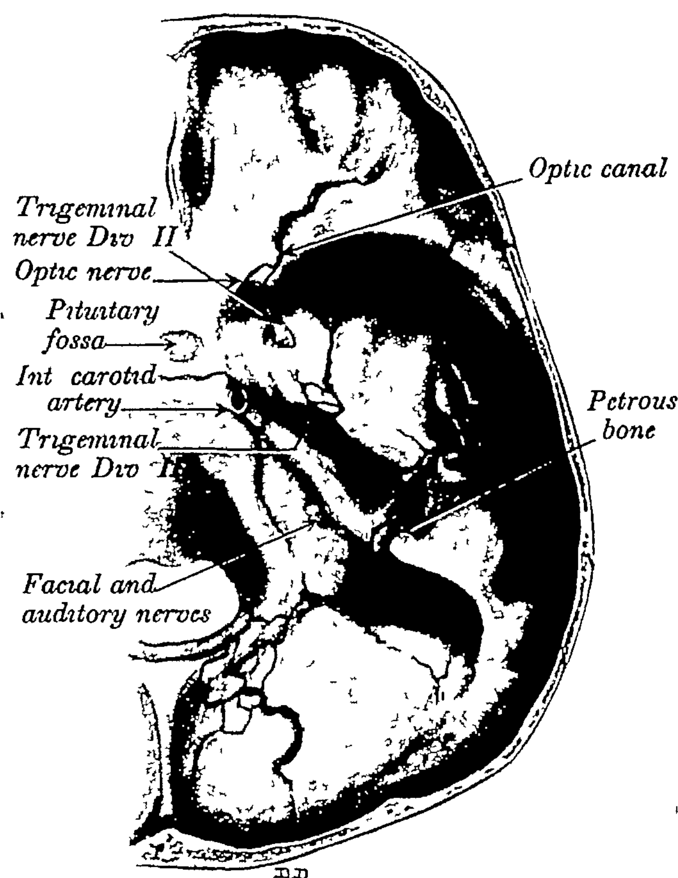


FIG 9

Such fracture patterns are probably due to a combination of injuring forces

INJURIES OF THE BRAIN

Apart from penetrating wounds of the skull the brain may be injured in one of two ways · (*a*) by distortions of the skull, or (*b*) by movements of the brain in relation to the skull as the head is thrown through space and then brought to rest In both circumstances a combination of injuring forces is active, as will be shown later.

In infants, while the sutures are still open and the bones plastic, the brain may be injured by distortion of the skull without a fracture occurring Alternatively, when the sutures are firmly closed and the bone has become brittle by calcification, any dis-

tortion of the skull sufficient to inflict an injury on the brain is usually of sufficient degree to produce a fracture. This means that cerebral injuries in adults which are unaccompanied by fracture must, in most cases, be due to movements of the brain within the skull.

From the tables in Chapter II it will be seen that the incidence of fracture of the skull in a clinical series of a thousand cases of acute head injury was as low as 68 per cent., whereas in fifty consecutive autopsies it was as high as 90 per cent. The difference of the fracture incidence in these two groups is due chiefly to the fact that fatal injuries of the brain are frequently caused by overwhelming violences which the cohesion of the skull cannot possibly resist

Movements of the Brain within the Skull.—When a man is thrown on his head against a resisting object the skull, at some phase of the injury, becomes stationary, whereas the brain, in virtue of its momentum, continues to travel onwards in the direction of the throw and is injured by those forces which resist its movement and finally bring it to rest.

The advancing surfaces of the brain come into forceful impact with segments of the skull or with the faces or edges of the dural septa, according to the circumstances of the accident. Because of their rigidity and their firm attachments to the bone the dural septa act as parts of the skull in resisting the movements of the brain

When the brain is thrown away from the base the upper halves of the outer surfaces of the hemispheres strike the vault of the skull; the upper surface of the corpus callosum strikes the free edge of the falx, and the upper surfaces of the cerebellar hemispheres are pressed against the under surface of the tentorium. In lateral movements the outer surface of one hemisphere strikes the side of the skull, whereas the inner surface of the opposite hemisphere strikes the flat surface of the falx; the brain stem impinges against the edge of the tentorium and one side of the cerebellum strikes the lateral wall of the posterior fossa. When the brain is travelling towards the anterior fossa in the longitudinal axis the anterior poles strike the anterior walls of the anterior fossa, the rostrum of the corpus callosum the edge of the falx, the brain stem the basi-occiput, and the anterior surface of the cerebellar lobes the anterior walls of the posterior fossa. In movements towards the base the under surfaces of the hemispheres and cerebellum are principally affected. Therefore according to the axis along which the brain moves, all combinations of surface injuries may occur, the nature of these depending on the speed at which the brain was travelling when its motion

was arrested and on the type of surface with which it made impact. For example, lacerations are particularly liable to occur when the brain is flung forcibly against the bony irregularities on the floor of the anterior fossa or against the sharp edges of the wings of the sphenoid bones

At the pole opposite to the site of impact the brain moves away from the skull, and before the space produced can be filled with cerebrospinal fluid a zone of diminished pressure results in which suction may be sufficient not only to rupture surface vessels

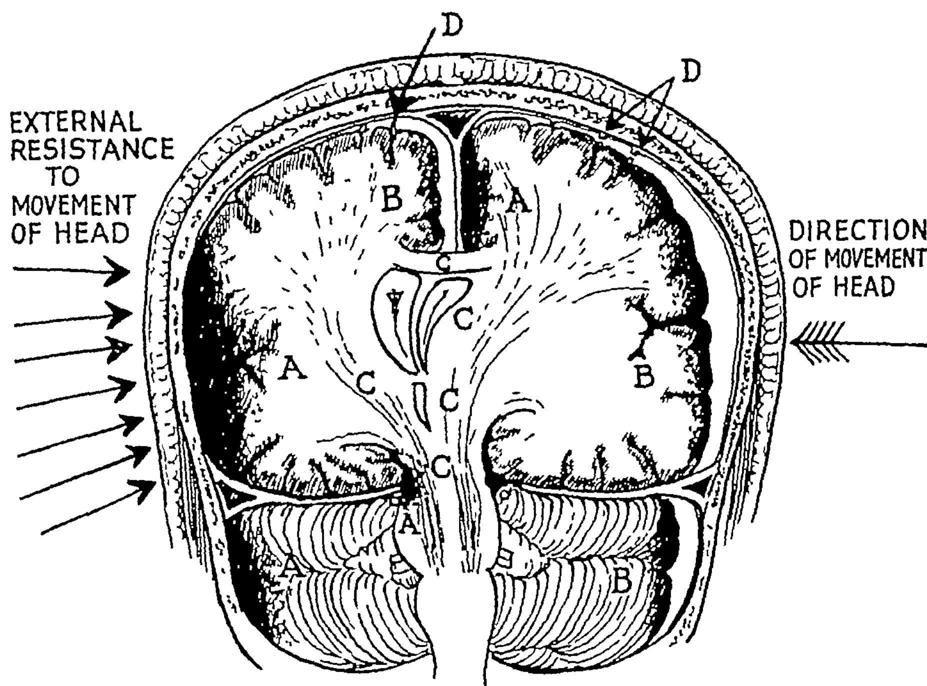


FIG 10

The mechanism of cerebral injury typical in road accidents

A, Injury by impact
B, Injury by suction
C, Injury by distortion
D, Rupture of vessels by stretching

but also those more deeply placed (Fig. 10) The importance of the part played by suction in cerebral trauma has recently been emphasised by Dott.[1]

Moreover, apart from changes in intracerebral pressure, the mere sliding of the brain within its dural coverings is a very serious happening, for not only does it account for the rupture of tethering blood vessels but also for the avulsion of cranial nerves The vessels which drain the cortical veins into the large venous sinuses have very fragile walls, and although they often run for half an inch or more in contact with the under surface of the dura, they are firmly attached to it; furthermore, their course across the

[1] Dott N M "Thompson and Miles Manual of Surgery," 1939, 2

subdural and subarachnoid space is short and straight. and hence they are easily torn when the cortex is displaced. It is rupture of these communicating veins which accounts for most cases of profuse subdural or subarachnoid hæmorrhages.

On the other hand, the large arteries at the base of the brain run a tortuous course, and a good deal of slack has to be taken in before they become taut. The tortuosity of the arteries and strength of their walls explain why they are so rarely torn (Fig. 11) When rupture does occur it leads to rapid death, except in

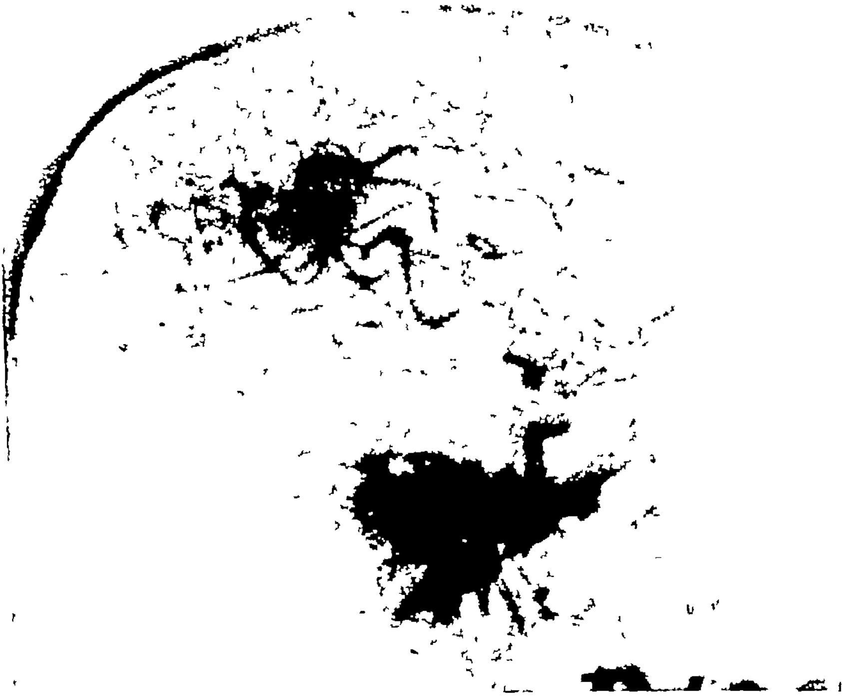


FIG 11

An angiogram depicting the vessels of the brain showing how a wide and safe range of movements is afforded to large cerebral vessels by their tortuosity

the rare instances when the internal carotid artery bleeds into the cavernous sinus, with a resulting pulsating exophthalmos. The smaller arteries which enter the base of the brain through the perforated spots or supply the optic chiasma are much more easily avulsed.

That the brain behaves as a homogeneous body of uniform consistency is a very erroneous impression. It is, in fact, composed of tissues of different specific gravity, toughness and tensile strengths. Moreover, it does not act as a single unit. Each hemisphere is separate from the other, apart from its relatively narrow connections through the corpus callosum and commissures, and both hemispheres are connected with the cerebellum only through narrow pathways of the brain stem This means that

large anatomical units of the brain can move in relation to each other, as well as the whole brain in relation to the skull, with the result that connecting pathways may easily be bent, stretched or torn whenever the brain is made to alter its shape. To a lesser extent the forces of distortion affect the connecting tissues between the white and grey matter and probably between every individual cell.

The brain can move within the skull in spite of the fact that its tissues cannot be compressed into a smaller volume, because it does not completely fill the intracranial cavity and because the cerebrospinal fluid which occupies the extra space can be displaced from one chamber of the skull to another or into the spinal theca, to accommodate cerebral displacements.

The cerebrospinal fluid also acts as a cushion to the brain and, in the mechanism of injury under consideration in this section, tends to minimise the injurious effects of cerebral movement, and particularly protects the advancing surfaces from laceration.

How much compression of the blood vessels at the site of impact leads to displacement of blood into extracerebral vessels is not known, probably not to a considerable degree, though sufficiently to cause some anæmia in the part concerned.

According to the circumstances of accident, injury of the brain by the mechanism of movement may be brought about by forces resulting either from deceleration or acceleration.

In the preceding section the results of forces due to deceleration have been discussed and it now remains to consider what happens to a man when he is set into rapid motion from a stationary position.

Let us suppose that he is standing on the road when he is struck by a car In a fraction of a second he is set into motion from zero to, say, thirty miles an hour or, in other words, he is struck by a force which causes tremendous acceleration. If a soft part of the body is struck it will give a little, thus reducing the rate of immediate acceleration and minimising the effect of the blow. If, however, the head is struck the resistant bone of the skull will not give in the same way as the soft tissues of the body, with the result that the rate of acceleration will be greater. In this case the head may be compressed against the spinal column and thus the brain may also be injured by the forces of skull deformation. If a man is carried forwards on the radiator of a car which gradually comes to rest and he is not thrown, then the forces due to acceleration only are concerned in the infliction of the injury. If, however, he is thrown forwards he may be brought to rest abruptly by his head striking an object which resists his motion,

and supposing he had been struck over a soft part of the body he may be brought to rest in a shorter time and over a shorter distance than he was set into motion. In this case the forces of deceleration may be greater than those of acceleration if the body has not lost considerable speed when the last impact occurs.

For a long time racing motor cyclists have realised how important it is to hold on to their bicycles when a crash comes so as to guard against the possibility of being thrown on to their heads and thus being injured by the forces of deceleration They know that after most collisions a bicycle will skid and come to rest relatively slowly.

Deformations of the Skull.—In distortions of the skull there is a lag in the moulding of the intracranial contents to the new curvatures of the bone, so that parts of the brain are subjected either to increased pressure or to suction, and no part of the brain escapes the effect of the injurious forces Also, as in the previous mechanism, the tissues of the brain are bent, stretched or torn as the result of distortion

Moreover, as the skull rebounds after the injuring force has ceased to act, important structures may be torn, and in particular, if the dura does not follow the movements of the skull the meningeal vessels will be pulled away from the bone and small nutrient vessels broken Rupture of the main vessels themselves depends on certain anatomical peculiarities Often the vessels are embedded in a deep bony groove, with overhanging edges in the proximal part of their course, whereas from the Sylvian point onwards they are firmly attached to the dura, and therefore will

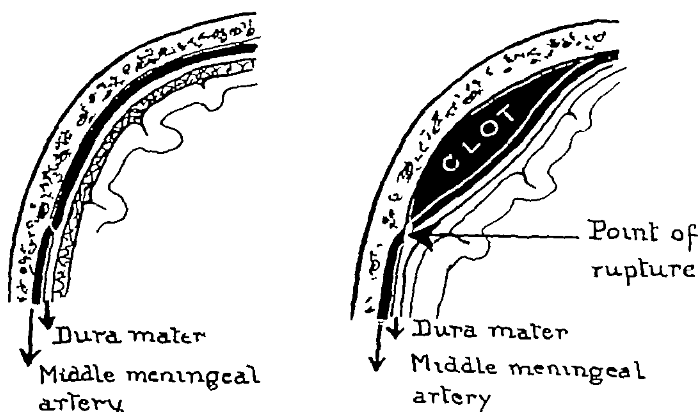


FIG 12

Rupture of the middle meningeal vessels by stretching at the point where they leave a bony tunnel or groove to become attached to the dura

break at this point of junction whenever the skull moves away from the dura (Fig. 12).

It has often been stated that damage of the brain can occur

by "contrecoup," but what is meant by this mode of injury has never been made very clear. Usually the term "injury by contrecoup" is used purely in the sense that a pole of the brain opposite to the site of impact has been damaged and not to indicate the particular physics by which the injury was produced (Fig. 13).

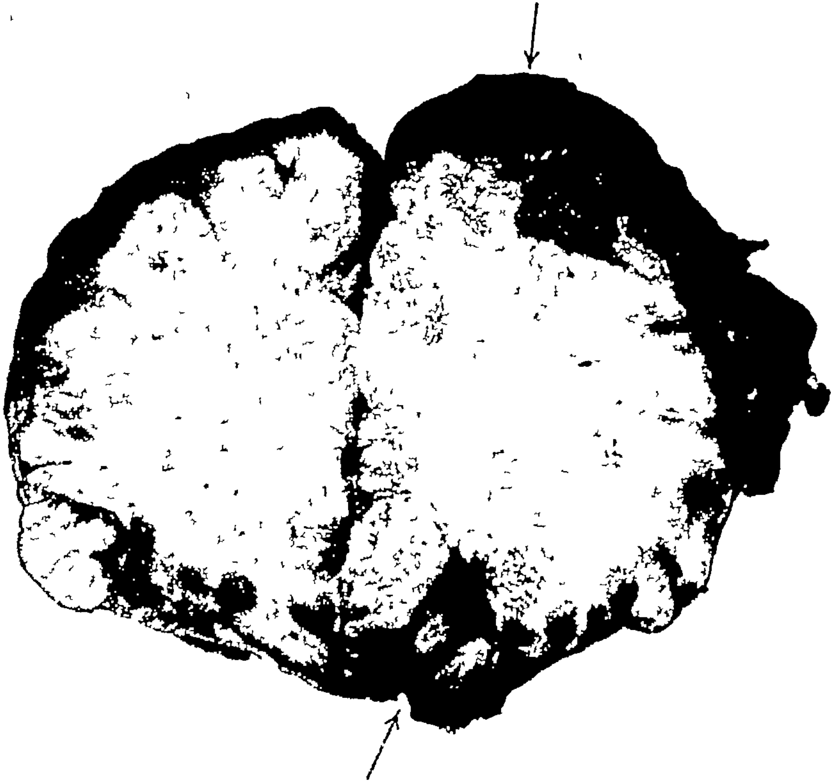


Fig 13
A good example of contrecoup injury of the brain

This limited implication of the term is probably the correct one, as cerebral lesions of a contrecoup distribution can readily be explained by one or other of the above mechanisms, and they are produced in three ways :—

(*a*) By suction, either when the brain is flung away from the skull when the motion of the head is abruptly arrested against a resistance, or in distortions of the skull

(*b*) By the brain being struck by a distant arc of the skull as it is flattened in a deformation

(*c*) By the brain being thrust against a dural septum or face of the skull when its opposite side is struck by a local inbending of bone

The severity of an injury to the brain consequent on deformity of the skull again depends on the circumstances of the accident. For example, let us consider what may happen to a man when

swimming around a ship at anchor. If the ship rises slowly on the swell of the tide and knocks against his head in the open sea, he will merely be pushed away with very little damage having been inflicted. On the other hand, if he is swimming between the ship and the quayside, the same movement of the ship, if he is trapped, will crush his head to pulp.

In actual practice among human beings repeated accidents of a stereotyped pattern do not occur. Whether cerebral injuries are more frequently caused by the forces associated with movements of the brain or with deformities of the skull is not known, but what is certain is that overwhelming types of injury to the brain are the result of forces continuing to act after the skull has been deformed and fractured.

The outstanding feature of any closed injury to the brain, whether this is due to distortion of the skull or to movements of the brain within the skull, is that the whole brain is subjected to the injuring forces Lacerations and contusions certainly constitute the more obvious type of lesion and show that parts of the brain have been more severely damaged than others, but they do not indicate that the damage has been restricted to these areas.

PENETRATING AND GUNSHOT WOUNDS

Penetrating Wounds in Peace Time.—In peace time penetrating wounds of the brain are rare, because the circumstances of the accident are such that a small mass does not often strike the skull at a sufficiently great speed to pierce the thick and tough bones of the vault. When they do occur they are usually due to indriven fragments of bone, and less frequently to pointed metal instruments or to such things as the spikes of iron railings. The type of force which causes a depressed fracture of the vault usually bursts the scalp and comminutes the bone, but even in those cases where the dura is pierced it is very rare for a bony fragment to become embedded deeply in the cerebral tissue, and rarer still for it to become completely buried.

The relatively thin panels of bone at the base of the skull for obvious reasons are not vulnerable to piercing types of injury, save at the roof of the orbit where a penetrating wound may involve the brain, but I have known this to happen in two instances only.

In one case a child fell from a tree on to a pointed stick, which travelled along the inner wall of the orbit without damaging the eyeball and penetrated the frontal lobe of the brain by passing through the floor of the anterior fossa. An uninterrupted recovery was made after the stick had been removed. The second

case happened in an air raid, when a piece of wood, blown into the orbit by the explosion of a bomb, ruptured the globe of the eye and became embedded in the sphenoidal fissure, causing a fatal meningitis. Examples have been encountered of people poking a way into the brain through the orbit or nose in an attempt to get relief from severe and intractable headaches. In one case I know of this led to the formation of a frontal abscess.

In operations at the roof of the nose for ethmoidal sinusitis, adenoids or nasal polypi, cerebral structures may be damaged on those occasions when a pre-existing intracranial lesion has led to destruction of the base of the skull. For example, I have known of a saccular aneurism of the internal carotid artery being ruptured, a pharyngeal meningocele being shaved off and the dilated third ventricle of a hydrocephalus being opened In each case, with appropriate treatment, the patient lived.

Air-raid Casualties.—In air-raid casualties lacerations of the scalp of all degrees are exceedingly common, whereas compound depressed fractures of the skull, and in particular penetrating wounds of the brain, are surprisingly rare No doubt this is due to the way the injury is produced, for a bomb fragment is usually a large, not a small, piece of metal, and its initial velocity is very great, so that if it strikes the head squarely it is likely to disintegrate it completely and cause instantaneous death Non-fatal penetrating wounds are due to small missiles, and often more than one fragment of metal is found deeply embedded in the cerebral tissue, and these at distant sites.

As far as can be judged, most cranial and cerebral injuries which occur in air raids are due to the indirect effect of blast. Either the patient is thrown on his head or his head is struck by falling masonry or by moving objects This means that the head is injured by forces of relatively low velocity, and it is those of high velocity which tend to penetrate the skull. A sufficiently heavy object, of course, even if travelling at a low speed, will completely crush the head and burst the scalp, but again this type of injury is fatal.

The direct effect of blast on the head is not known with any certainty, but from the power that it may exert up to many hundreds of tons per square inch it is obvious that all degrees of cerebral and cranial injury are possible, and on occasions the head is blown to bits Stewart, Russell and Cone,[1] basing their opinion on the correlation of the clinical signs and post-mortem findings on a stunned pheasant which they found at the edge of

[1] Stewart, O W, Russell, C K, Cone, W V "Injury to central nervous system by blast Observations on a pheasant" *Lancet*, 1941, **240**, 172

a bomb crater, came to the conclusion that as a result of acute thoracic compression a column of blood may be driven up the vessels of the neck forcibly enough to cause concussion or intracerebral hæmorrhage.

Gunshot Wounds.—Gunshot wounds of the head have aroused interest from the time small firearms were introduced many centuries ago, and it was not surprising to find in the literature that extensive researches were made on this subject by the Imperial Royal Prussian War Department,[1] both on the living and on the cadaver. At the beginning of the last war what was virtually the textbook of military service was written by Sir George Makin [2] on his "Surgical Experiences in South Africa," and it makes extremely interesting reading. When in 1922 the history of the medical services of the 1914-18 war was published, the subject of gunshot wounds of the head was reviewed by Wagstaffe,[3] and no doubt at the end of this war new and important chapters will be written.

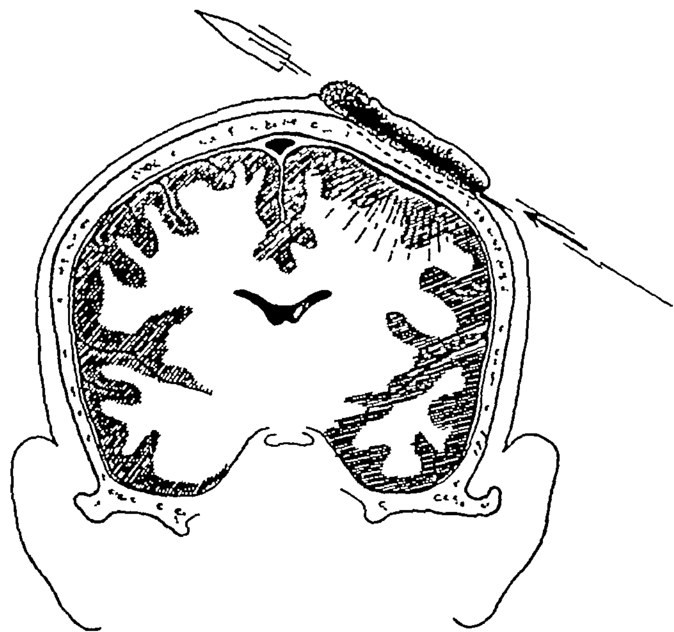


FIG 13A

Local percussional violence

Exemplified by tangential bullet wound of scalp and skull The skull is locally and momentarily displaced by the blow The bending in is of small extent but at high velocity The underlying brain is locally percussed and its function temporarily deranged (*Norman Dott*)

The varieties of gunshot wounds of the head are legion. All degrees of injury from metallic staining of the outer table of the skull to complete shattering of the head have been described, and Cushing [4] classified the wounds according to their depth. The type, extent and depth of a wound depend on the site at which the skull is struck, on the angle of striking, on the physical properties of the skull, on the speed of the bullet at the time of

[1] Coler, A G E von, Schjerning, O von "Ueber die Wirkung und die kriegschirurgische Bedeutung der neuen Handfeuerwaffen" *Int Congr Med, XI, Rome*, 1894-95, **4**, Chir 232

[2] Makin, Sir George "Surgical Experiences in South Africa"

[3] Wagstaffe, W W "Official History of the War" 1922 *Med Serv Surg of the War*, **2**, chapter i

[4] Cushing, H "Study of a series of wounds involving the brain and its enveloping structures" *Brit Jour Surg*, 1917-18, **5**, 558

impact and on whether or not the bullet changes its shape after impact.

According to Wagstaffe a bullet which strikes the head tangentially may cause a disturbance in the brain sufficient to produce concussion without fracturing the skull or lacerating the scalp, but this type of injury is rare (Fig. 13A). Usually a bullet or piece of shrapnel strikes the head more or less squarely and

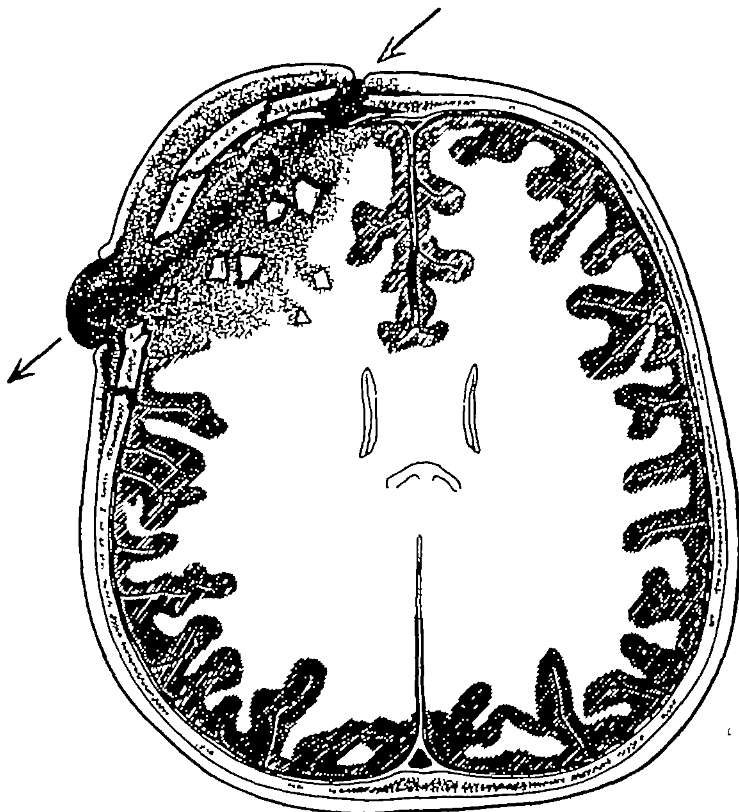


FIG 13B

Violence by high velocity penetrating missile

A missile has passed through head as indicated It has produced penetrating fracture of skull at entrance and exit and has scattered fragments of bone along and around its track in brain A bursting fracture of skull has been produced by explosive effect of velocity A considerable volume of brain tissue has been disintegrated around track through brain Hæmorrhage is causing accumulation of clots and extrusion of damaged brain matter through external wounds (*Norman Dott*)

causes a penetrating wound which, of course, is necessarily compound (Fig. 13B).

Individual and racial differences in the thickness and brittleness of skulls are considerable, and when the injuring force is not of overwhelming magnitude they may determine whether a serious or a simple injury results. For example, thin skulls of children may be pierced by a bullet at the end of its flight when it is coming to rest, whereas the skulls in some types of negro are so

thick and tough that they have been known to hold up a bullet fired at point-blank range.

Dumdum or soft-nosed bullets which easily flatten on impact, lacerate the tissues much more severely than do "full-jacketed" or hard-nosed bullets which do not lose their shape and



FIG 14

A large piece of metal lies at the bottom of the wound track, whereas smaller metallic and bony fragments (*a*, *b*, *c*) are deposited superficially

when fired from a short distance have almost the same result as a local explosion, their shattering effect is so great.

At any point on its trajectory the force a bullet possesses depends on its momentum, which is the product of its mass and velocity. Therefore, as the mass of the bullet is a constant, and as the bullet gradually loses speed from the moment it leaves the gun, the damage it is able to inflict on the head, all other factors

being equal, must be proportional to the distance from which it was fired Experiments made on the cadaver by German investigators, with a rifle firing a hard-cased bullet at a velocity of 2,000 ft. per sec , showed that a bullet readily passed through the head and did not become arrested in the brain tissue until a distance of 8,700 ft. was reached The amount of damage a modern rifle bullet is capable of doing can be judged from its ability to pierce a brick wall 21 in. thick at battle range

When a bullet traverses the intracranial cavity it not only destroys the structures in its pathway but also produces an expansile or explosive effect, the forces of which are transmitted throughout the brain tissue and cerebrospinal fluid. The explosive effects of a bullet fired from a short distance may be sufficient to burst the scalp, shatter the skull and dislodge the brain Occasionally the whole or mangled brain is blown a distance of many yards, and this happening is called " exenteratio cranii." In the less severe types of injury a cone of damaged cerebral tissue is all that surrounds the wound track, and between the two extremes all degrees of cerebral and cranial injury may happen

A point of interest, as well as one which illustrates the mechanism of bending in producing a fracture, is that the inner table at the entrance hole of a bullet wound is always much more extensively comminuted than is the outer table, whereas the opposite happens at the exit hole

Of the severe types of injury the one which concerns the surgeon particularly is where the bullet remains in the intracranial cavity. In these cases pieces of bone and occasionally pieces of hair or cloth are carried inwards with the bullet and scattered in the superficial part of the wound track, whereas the heavier missile becomes buried deeply in the cerebral tissue, and thus two widely separated foci of possible infection are introduced, either of which may lead to abscess or spreading encephalitis (Fig. 14).

CHAPTER II

PATHOLOGY

INTEREST in disease of the brain started, as far as is known, in the neolithic age many thousands of years ago, when holes were rubbed into the skull by flints to let out those demons which to-day would be designated by more formal pathological terms

Actual reference to injuries of the skull was made as early as 1600 B.C. in a papyrus [1] written by a surgeon who knew that the pulse may be affected by a head injury, that the patient may sustain a depressed fracture of the vault even though the scalp was not torn, and that such injury, if it involved the brain, might cause loss of speech and paralysis of the limbs. Hippocrates [2] chronicled his experiences on wounds of the head in the fifth century B.C. and in his discussion on operative treatment warned the surgeon not to injure the brain, otherwise convulsions and palsy would ensue. This advice no doubt was found useful later by Galen, for whom Roman arenas provided a great number of cases on which to practise the art of trepanning. In A.D. 1170, Roger of Palmero [3] published his " Practica Chirurgica," of which the first part is concerned with wounds of the head and fractures of the skull. He advised that a finger should be introduced into open wounds and moved over the underlying bone so as to discover the presence of a fracture, and this practice is still in use to-day. Since those early days a voluminous literature on cerebral and cranial injuries has collected and advanced in scientific importance as more accurate methods of observation were discovered, such as microscopy by Jansen in 1590. The correlating of clinical signs with post-mortem findings became firmly established at the end of the eighteenth century, and this practice, combined with newer experimental methods, led to great additions to knowledge by a succession of brilliant neurologists in the following century. The main problems of cerebral trauma, however, have by no means been solved in spite of the interest they have aroused, and to-day circumstances are making the

[1] Garrison, F H " History of Medicine," 55 Philadelphia, 1929

[2] Hippocrates " Wounds of the Head " Translation in Loeb Classical Library, 1927, 3. Edited by Capps, Page and Rouse

[3] Garrison, F H " History of Medicine," 152 Philadelphia, 1929

subject the concern of everyone engaged in traumatic surgery or medicine, not only because of the abundance of material but also because of its widespread distribution.

Between 8,000 and 9,000 people are killed each year on the British roads, and of these a large percentage die as the result of an injury to the head. Furthermore, for every fatality due to cerebral trauma at least four other people receive non-fatal injuries to the skull or brain which lead to prolonged morbidity.

ROAD ACCIDENTS

Year	Killed	Injured	Total
1927	5,329	148,575	153,904
1936	6,561	227,813	234,373
1937	6,633	226,402	233,035
1938	6,648	226,711	233,359
1939	8,272		
1940	8,609		

The number of people killed on the roads in Britain during the first three months of 1941 was 2,264
These are Ministry of Transport figures

These are important facts, and in order to give further perspective to the subject the relevant details of a clinical series of 1,000 cases and the pathological findings of fifty consecutive post-mortem examinations will be found in tabulation form below.

A THOUSAND CONSECUTIVE CASES OF ACUTE INJURY OF THE HEAD

Age in Decades	Sex.		Fractures with the Scalp Intact.			Compound. Fracture of the Vault.		Associated Injuries.				Type of Accident.			Number of Deaths	
	M	F	X ray Evidence of Fracture	Clinical Evidence of Fracture.	No Evidence of Fracture.	Linear	Depressed.	Spine	Limbs.	Thorax.	Abdomen.	Road	Industrial.	Other Types.	M	F
1	84	48	57	35	42	3	6	0	16	6	0	72	0	60	7	4
2	160	36	82	32	74	5	7	0	18	8	0	144	38	14	19	7
3	172	32	75	50	73	6	8	0	12	6	0	136	28	40	24	5
4	112	44	75	30	51	8	5	2	10	4	2	84	52	20	12	9
5	80	28	66	22	16	9	3	2	14	2	2	68	28	12	15	6
6	48	48	52	21	25	3	2	0	4	0	1	48	24	24	11	11
7	44	20	37	17	17	1	0	2	12	2	0	40	8	16	9	7
8	24	20	28	7	16	2	0	2	6	4	0	20	0	24	8	9
TOTAL	724	276	472	214	314	37	31	8	92	32	5	612	178	210	105	58
Percentages	72 4	27·6	47 2	21·4	31·4	3 7	3 1	0 8	9 2	3 2	0 5	61 2	17 8	21·0	10 5	5 8

NOTE —*This series does not include war or air raid casualties.*

The incidence of head injuries in air-raid casualties has not yet been published, but the following is an example from my own experience.

Approximately 192 people were in an air-raid shelter when it received a direct hit. One hundred and five people were killed instantly. Between 30 and 40 people received injuries which necessitated surgical treatment; of these, 22 received injuries to the head, 7 of which were serious wounds, the dura being penetrated in four cases.

NUMBER AND TYPE OF OPERATIONS PERFORMED IN THE ABOVE SERIES

Type of Operation	Recovered	Deaths
For extradural hæmorrhages	9	2
For acute subdural hæmorrhages	2	3
For chronic subdural hæmorrhages and hygroma	3	0
Decompressions for raised intracranial tension	5	2
Exploratory trephine openings	10	3
For compound depressed fractures	14	7
Excisions of compound linear fractures	5	2
To raise depressions in closed fractures	3	0
Total number of operations	51	19

NOTE —This list does not include repairs of simple wounds of the scalp which were multitudinous Spinal drainages or other methods of dehydration are also omitted, as these were routine measures in many of the cases

Two meningeal hæmorrhages not operated on were found at autopsy —

(a) One was diagnosed but died before operation could be performed

(b) One was not diagnosed because there was no latent interval This was complicated by a profuse subarachnoid hæmorrhage

CAUSES OF DEATH IN ABOVE SERIES

Cause of Death.	Within Twelve Hours.	Within Twenty four Hours.	Second Day	Third Day	Fourth Day	Fifth Day	Sixth Day	Seventh Day	Second Week	Third Week.	Later	Total No. of Deaths due to each Cause	Percentage of Deaths due to each Cause
Concussion or decompression	47	54	15	9	1	2	1	2	2	1	0	134	82·0
Meningitis	0	0	1	1	2	0	0	0	3	1	1	9	5 5
Pneumonia	0	0	2	2	3	1	0	0	1	0	2	11	06 7
Associated Injuries	2	0	3	1	0	0	0	0	0	0	0	6	3·6
Exhaustion	0	0	0	0	0	0	0	0	0	2	0	2	1·0
Loss of blood	0	0	0	0	1	0	0	0	0	0	0	1	0·6
Total number of deaths in 1,000 cases	49	54	21	13	7	3	1	2	6	4	3	163	
Percentage of deaths in 1 000 cases	4 9	5·4	2 1	1 3	0 7	0 3	0 1	0 2	0·6	0·4	0 3		

Percentage fatality rate, 16 3

FIFTY CONSECUTIVE AUTOPSIES

	Brain Tissue Undamaged Macroscopically	Lacerations		Surface Contusions		Intracerebral Hæmorrhages.		Subarachnoid Hæmorrhages	
		Superficial	Deep	Single	Multiple	Petechial	Massive	Slight	Profuse
Number of cases in which each lesion occurred	2	9	5	18	24	23	1	9	27
Percentage occurrence	4	28		84		48		72	

	Acute Subdural Hæmorrhages.		Extradural Hæmorrhages		Meningitis.	Fractures of the Skull				Associated Injuries
	Slight	Profuse	Thin Layer	Large Clot.		Vault	Base	Both	Neither	Severe
Number of cases in which each lesion occurred	3	5	7	3	3	5	4	36	5	15
Percentage occurrence	16		20		6	10	8	72	10	30

This group was recently collected from all sources and includes material partly from outside clinics and partly from the later fatalities of the above clinical series.

In the two cases in which no damage occurred to the brain tissue, extensive surface hæmorrhages were found—one extradural and the other subdural. Surface contusions occurred most commonly on the under and lower parts of the outer surfaces of the hemispheres and frequently in the cerebellum. Severe lacerations were usually found under displaced fractures, whether these were open or closed. In one case the hypothalamus had been severed transversely. The most profuse surface hæmorrhages were due either to tearing of the parasaggital sinus or to the veins which drain into it

There were ten cases of extradural hæmorrhage. Of these, seven were thin clots and, judging by the severity of lesions elsewhere in the body, were of little significance. In three cases a massive clot was found · one, due to basal rupture of the meningeal vessels, was uncomplicated by lesions elsewhere in the brain , the others—one due to tearing of the parasaggital sinus and the other to diploic bleeding—were complicated by other cerebral lesions Fracture of the skull occurred in 90 per cent of cases and in 72 per cent both the base and vault were involved. When the skull was not fractured, contusional lesions of the brain were not as pronounced as in fracture cases, but surface hæmorrhages were equally profuse. Severe injuries elsewhere in the body were common. Subgaleal bleeding was also common.

THE PATHOLOGY OF CLOSED INJURIES OF THE BRAIN

The Three Primary Pathological States

During the few moments the accidental forces are operating the brain can be damaged in three and in only three ways. It may be contused or lacerated or its neurones may undergo a diffuse neuronal injury of submacroscopical dimensions. One or any combination of these conditions may occur. All other phenomena such as œdema and massive hæmorrhages are secondary, even though they may develop immediately afterwards.



Fig 15

A small contusion on the under surface of the right cerebellar hemisphere caused by movement of the brain within the skull

Contusions.—Cerebral contusions are microscopical solutions of continuity of brain tissue. They may occur on the surface of the brain or deep within its tissue, and both types are necessarily associated with bleeding though this is usually punctate and limited to the area of the bruise When superficial they are seen as subpial gelatinous-looking stains of a mottled reddish-blue colour covered by a thin layer of blood (Fig. 15) Thick clots cannot occur because the pia is firmly attached to the brain tissue and will not readily strip. Often contusions are wedge-shaped with the apex of the wedge extending into the cerebral tissue for a distance of 1 or 2 cm., but rarely deeper than

this.[1] They may be single or multiple; any surface or more than one surface of the brain may be involved, including the upper aspect of the corpus callosum, and they may be no bigger than a pinhead or may cover large areas on both sides of the brain.

Within the brain substance, contusions are seen as clusters of petechial hæmorrhages superimposed on a dull grey background. Most commonly they occur in the white matter of the hemispheres, but they are also found in the cerebellum (Fig 16), medulla, pons,[2] midbrain, thalamus, hypothalamus, caudate nucleus, lenticular nucleus and in the subependymal layers of the ventricles and of

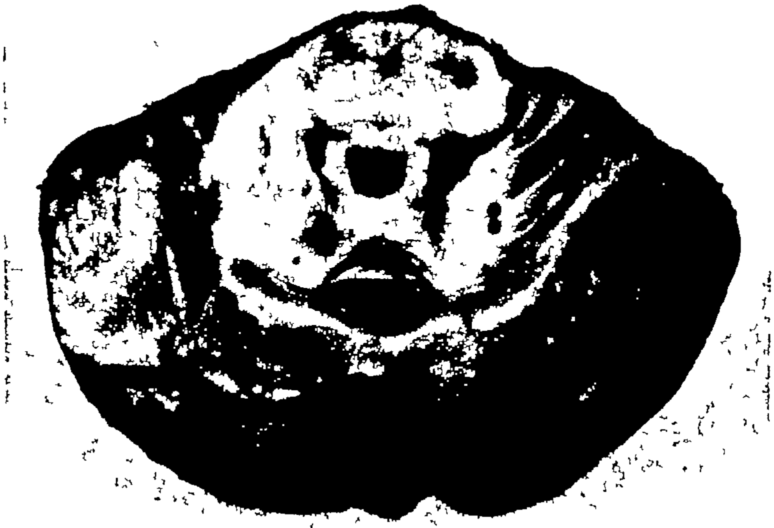


Fig 16

Petechial hæmorrhages within the brain stem and cerebellum

the aqueduct of Sylvius They may be single or widespread in distribution.

Microscopically, either superficial or deeply placed contusions show as spots or streaks of extravasated blood surrounded by an area of damaged brain cells, beyond which is a limited zone of œdema [3] The extravasated blood is due either to ruptured vessels or diapedesis The area of damaged brain cells is composed of three concentric zones, each one being distinguishable from the

[1] Courville, C B "Pathology of the Nervous System," Part IV, 223 Pacific Press, California, 1937

[2] Greenfield, J G "Some Observations on Cerebral Injuries" *Proc Roy Soc Med*, 1938, **32**, 43-52

[3] Rand, C W, and Courville, C B 'Histologic Studies of the Brain in Cases of Fatal Injury to the Head (6) Cyto architectonic Alterations" *Arch Neur and Psych*, 1936, **36**, 1277-1293

other by the nature of the cellular change within them. Nearest to the extravasated blood the neurones, glia and microglia are completely destroyed and often are in a state of liquefaction. In the middle zone the staining properties and shapes of the cells are altered in such a way that it is impossible to know whether the cells have been permanently injured or not. At the periphery the neuroglial astrocytes and the microglial cells are hypertrophied or increased in numbers, and it is from this zone of increased cellular activity that the processes of phagocytosis and repair are initiated.



Fig 17
A laceration of the outer surface of the right temporal lobe associated with a subarachnoid hæmorrhage

Many of the cells within a contusion, though functionally paralysed, are viable and will recover under favourable conditions or succumb if the cerebral circulation is impaired by secondary development such as œdema

Laceration.—Lacerations are gross solutions of continuity of brain tissue and differ from contusions merely in their severity. Most commonly they are found on the under surfaces of the frontal lobes and near the tips of the temporal poles (Fig. 17). Usually they are superficial, but may be deep enough to open into a ventricle or cut across a basal nucleus such as the hypothalamus. Deep lacerations are invariably associated with displaced fractures and may occur anywhere. Contusions of the choroid plexuses and walls of the ventricles are common. Lacerations of these structures, on the other hand, are rare.

The pia is invariably torn in a laceration, which means that ruptured cortical vessels may bleed with little restriction into the voluminous subarachnoid spaces Tearing of the arachnoid membranes also is usual and results in blood and cerebrospinal fluid leaking into the subdural space The tough dura, however, rarely gives way and this minimises the incidence of post-traumatic epilepsy, as will be seen later.

Shaggy-walled cavities distended with blood are found in severe injuries, and occasionally whole lobes of the brain may be so thoroughly disintegrated that the tissue may be washed away with jets of water. In severe crushing injuries complete pulping of the brain may occur.

The Healing of Contusions and Lacerations.—The processes of repair in the brain are similar in principle to those elsewhere in the body, inasmuch as dead tissue has to be removed and the wound consolidated. The histopathological details, however, are peculiar to the specialised cerebral tissues, and it is of interest to know what the possible final results of a wound may be.

There are three types of cell in the brain (Fig. 18) :—

(i) The neurones }—of ectodermal origin.
(ii) The neuroglia }
(iii) The microglia—of mesodermal origin

The neurones are the cells which initiate and transmit the nerve impulses When they die it is believed that they are never replaced either from embryonal sources or by division of a neighbouring cell, although it is possible for a neurone to recover its function after a period of prolonged inactivity.

The neuroglia is an interstitial tissue, but none the less a fundamental part of the nervous system, and together with the blood vessels supports the brain and gives it cohesion. Probably it is essential to neuronal metabolism. It is composed of two types of cells, the astrocytes and oligodendrocytes. The astrocytes are stellate in shape and have dendritic processes issuing from their angles, one of which, known as the sucker foot, is longer and thicker than the others, and is attached to the wall of a blood vessel. The oligodendrocytes are round or oval cells containing large nuclei which almost completely fill their protoplasm and their processes are fine and branched From their arrangement along the axons of the nerve cells it is presumed that they are principally concerned with the metabolism of myelin.

The microglia was so named by Hortega,[1] who proved that certain interstitial cells were neither astrocytes nor oligodendrocytes and that they possess migratory and phagocytic powers. In

[1] Rio-Hortega, P Del "The Microglia" *Lancet*, 1939, 1, 1023

the resting phase these cells have a dark nucleus surrounded by scanty protoplasm, and possess two or more slender processes which are feathery in appearance owing to short side branches. Presumably they are part of the reticulo-endothelial system and play an important rôle in all inflammatory and necrotic processes of nerve tissue

The first process in repair is liquefaction of dead tissue and extravasated blood cells, and the various stages of this activity may be seen in a contusion as it changes in colour from dark red

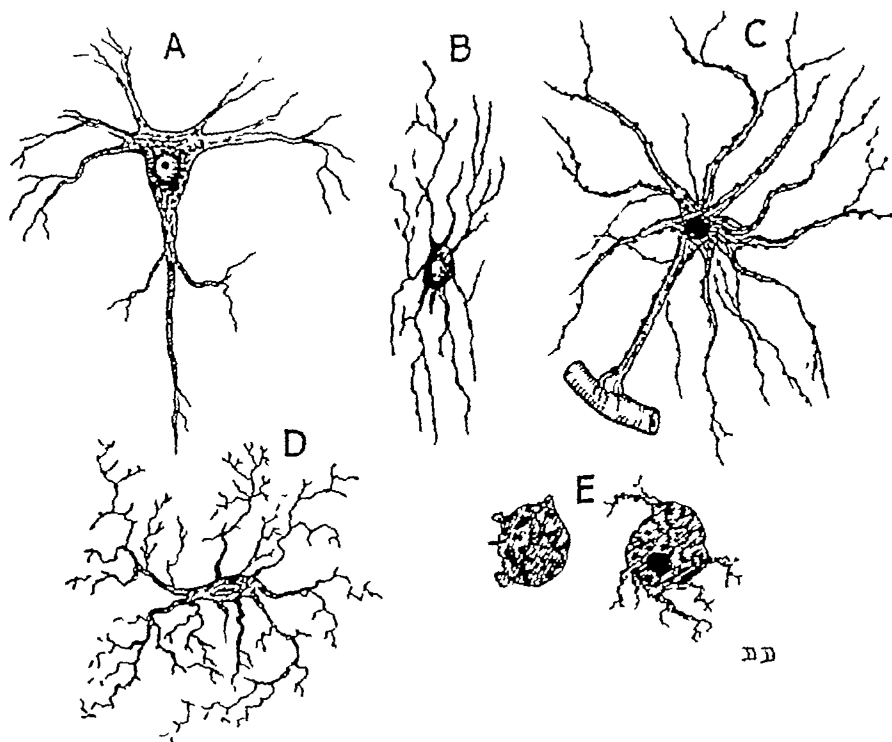


Fig 18

The types of brain cell A, Neurone, B, oligodendrocyte, C, astrocyte, showing sucker foot, D, microglial cell in resting phase, E, microglial cell in active phase (Gitterzellen)

through lighter shades of brown to a yellowish tinge. Physico-chemical phenomena account for liquefaction, and it has been suggested that the chemical agents concerned may come either from the damaged cells themselves or from the blood stream

At an early stage microglial cells from the surrounding brain tissue invade the damaged area and engulf the lipoids produced by the liquefaction, finally depositing them in neighbouring blood vessels or in the subarachnoid spaces When actively engaged in phagocytosis the cells change their shape, their processes are retracted and their bodies are swollen and granular. When in this condition they are known as scavenger cells, compound granular corpuscles or "Gitterzellen," and are most numerous on the sixth day following an injury.

According to Linell,[1] hypertrophy of the astrocytes surrounding a contusion is evident at the end of three days, and in three weeks these spider cells have migrated in considerable numbers into the wound. This condition is known as gliosis. In four weeks, felting of the astrocytic processes has occurred and in two months cell bodies have disappeared and a firm glial scar remains, the extent of which depends on the amount of dead tissue which had to be removed. A clean-cut wound, for example, as Penfield[2,3] has

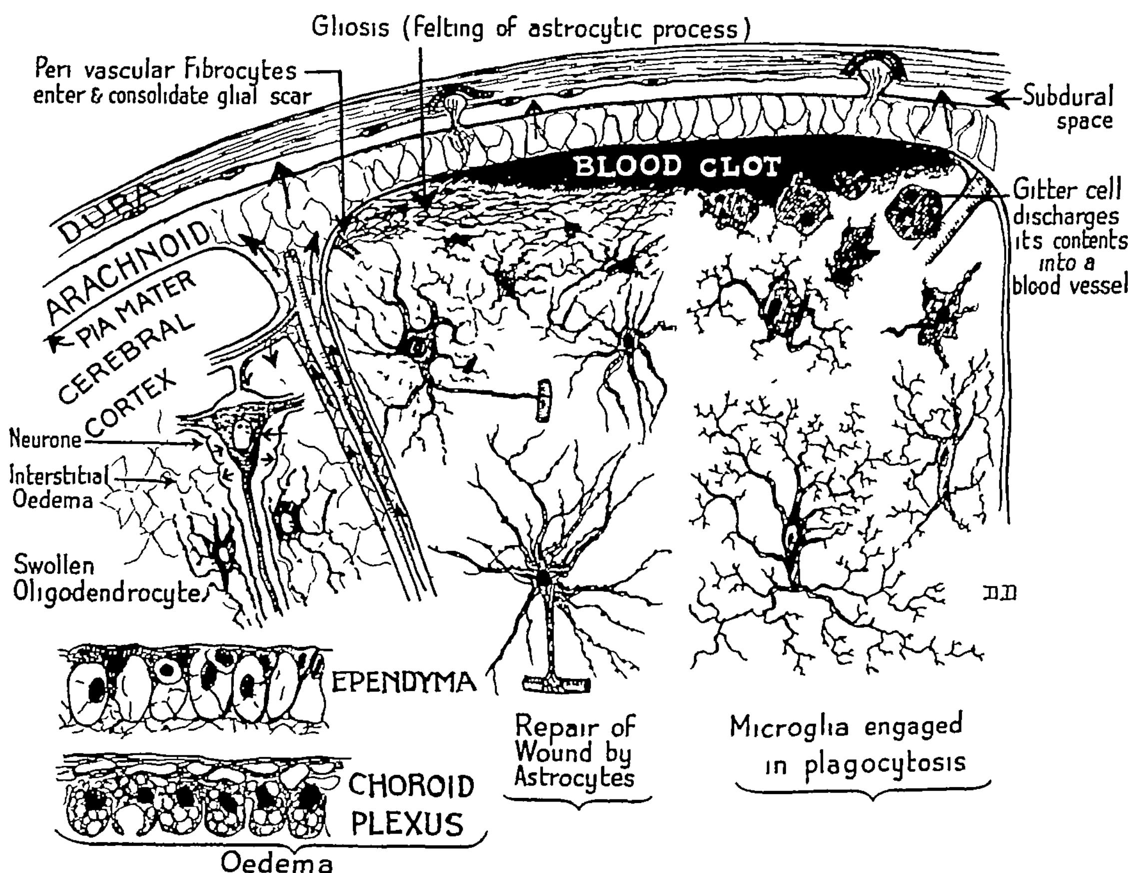


FIG 19

Repair of a cerebral wound On the left side of the picture is shown an area of œdema The perineuronal and perivascular spaces are distended with fluid and the cells of the ependyma and choroid plexus are enlarged and vacuolated The uncharted arrows indicate the circulation of the tissue fluids

shown, will heal with less gliosis than a lacerated wound with ragged edges. In simple contusions where there has been no gross loss of cerebral tissue the final healing may be obtained purely by glial processes. In lacerations or in contusions where there has been gross loss of tissue, the final cementing of the

[1] Linell, E A "Histology of Neurological Changes following Cerebral Trauma Experimental Investigation" *Arch Neur and Psych*, 1929, **22**, 926 948

[2] Penfield, W "The Mechanism of Cicatricial Contraction in the Brain" *Brain*, 1927, **50**, 499

[3] Penfield, W, and Buckley, R "Punctures of the Brain The Factors concerned in Gliosis and in Cicatricial Contraction" *Arch Neur and Psych*, 1928, **20**, 1

wound is brought about by fibrous tissue which grows in from the mesodermal elements of neighbouring blood vessels and particularly from those on the surface of the brain (Fig. 19) The manner in which fibrous tissue grows in from the surface explains why so



FIG 20
Severe cortical atrophy following trauma

many scars are attached to the leptomeninges In those cases where the loss of cerebral tissue has been great, cysts lined with fibrous tissue and containing xanthochromic fluid may be found within the gliocicatrix, and these no doubt are nature's economical way of filling in space.

A scar may or may not be the final result of a contusion or

laceration Occasionally a degeneration of the nervous elements takes place after an interval of a few weeks or even years, which proceeds far beyond the limits of the original contusion and leads to all kinds of neurological disorders. Also progressive softening



FIG 21

This is the type of closed fracture in which the meninges are torn and cerebrospinal fluid escapes into the subgaleal space to produce a traumatic meningocele It is also the type of fracture which acts as a natural internal decompression, as it allows blood to escape from the extradural into the subgaleal tissues, thereby avoiding compression of the brain It is the type of fracture most frequently seen in children, and explains how extensive calvarial fractures often occur without signs of severe cerebral compression

may result in delayed intracerebral hæmorrhage. This happening was described by Bollinger[1] as late traumatic apoplexy, and is

[1] Bollinger, O *Internationale Beitrage zur Wissenschaftlichen Med Festschr* Rudolf Virchow's Vollendung seines 70 Lebensjahres, 2 Berlin, 1891

of particular importance when considering the possible relationship of a sudden cerebral crisis to previous cerebral trauma.

The condition of "punch drunk" is well known in professional boxers and is due to degenerative changes in the brain consequent on small repeated hæmorrhages or contusions which, in themselves, are of no particular importance.[1,2]

In syphilitics or arteriosclerotics a degenerative encephalopathy may be precipitated by an injury, or a pre-existing diseased state may be aggravated and its pathological processes accelerated. Cortical atrophy is often the final result of a severe injury to the brain as proved by encephalography done in the routine investiga-

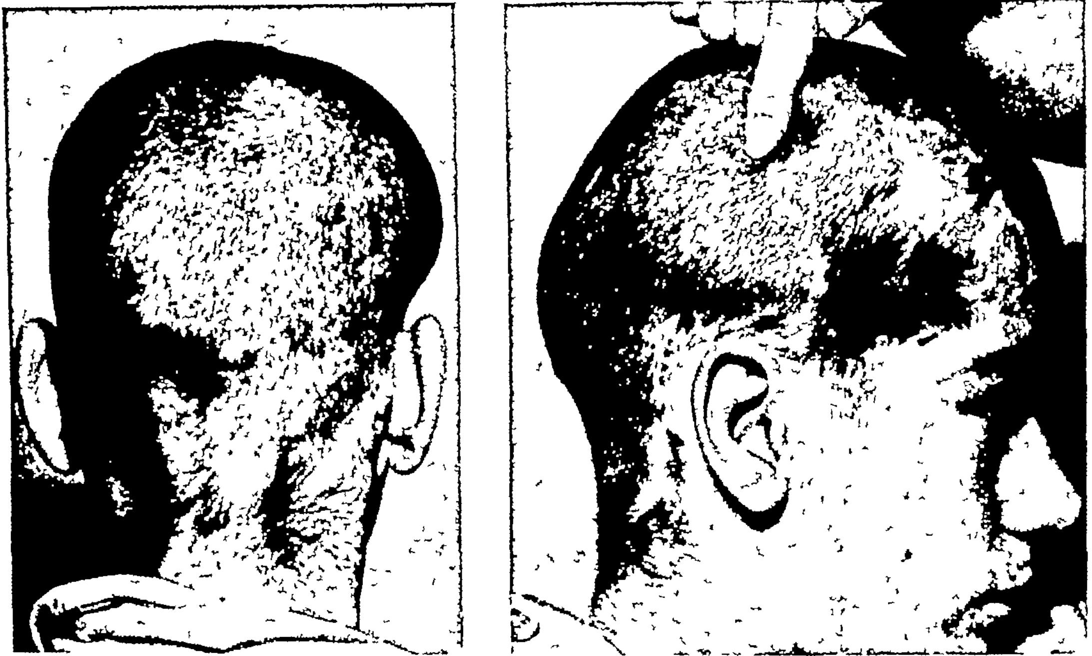


Fig 22

The beginning of a traumatic meningocele The swelling on the right side of the head is due to an escape of bloodstained cerebrospinal fluid beneath the scalp

tion of post-concussional symptoms, when the air studies show that the loss of tissue is compensated for by dilatation of the lateral ventricle which appears to wander into the hemisphere concerned (Fig. 20).

On those rare occasions when a closed linear fracture is associated with a tear in the underlying dural and arachnoid membranes, cerebrospinal fluid escapes into the subgaleal space, and a cyst communicating with the subarachnoid cavity may develop beneath the scalp (Figs. 21 and 22). Such a condition is known as a traumatic meningocele and occurs more commonly in children, possibly because the firmer attachment of the dura

[1] Martland, H S "Punch Drunk" *J A M A*, 1928, **91**, 1103

[2] Parker, H S "Traumatic Encephalopathy (Punch Drunk) of Professional Pugilists" *J Neur and Psych*, 1934, **15**, 20

to the bone in the young renders it more liable to tearing than its looser attachment in adults. The wall of the meningocele is non-absorbable and results from reaction of fibrous tissue cells lining the scalp to the irritation of the cerebrospinal fluid. Such reaction in non-neural tissues to the presence of cerebrospinal fluid always occurs and is a manifestation of the inherent tendency



Fig 23
A case of traumatic malacia or absorption of bone following trauma The scalp was not broken at the time of injury

of the nervous system to insulate itself This tendency to neural insulation is of great surgical importance, as it explains the failures of operations designed to relieve hydrocephalus by drainage of the cerebrospinal fluid into the subcutaneous or submuscular spaces. When an opening through the dura and skull is wide, or when large areas of bone disappear owing to malacia (Fig. 23), brain tissue may herniate into the meningocele and convert it into a meningo-encephalocele or into a meningo-encephalo-ventriculocele if the ventricle is also included in the brain hernia.

DIFFUSE INJURIES

Contusions, lacerations and hæmorrhages commonly found at autopsy, or on surgical exploration after acute head injuries, often will not account for unconsciousness with its allied neurological states. Therefore cells or pathways concerned in the initiation or transmission of nervous impulses other than those in the areas which have undergone damage of macroscopical dimensions must have been affected by the injury. The actual nature of the changes in the neurones concerned constitutes one of the main problems of acute cerebral trauma and, in view of the great controversy the subject is arousing, it is better to avoid the use of terms such as commotio cerebri, which imply that the physiopathology of diffuse traumatic neural paralysis not due to gross contusion or compression is known. The term "concussion," although sometimes used to mean that the brain tissue itself has been concussed—a hypothetical form of injury—is best restricted to a clinical sense denoting that the state of consciousness has been impaired as the result of a mechanical force applied to the head. Much experimental work has been carried out in an attempt to elucidate the neuropathology and neurophysiology of concussion, and to-day there are three main theories.

The Vascular Theory.—Trotter,[1] basing his ideas on the work of Kocher,[2] believed that in acute distortions of the skull the capillaries of the brain are obliterated by compression, and he suggested that the resulting cerebral anæmia accounted for unconsciousness. The sequence of neurological events in non-fatal cases he ascribed to the gradual recovery of the circulation, which goes through a phase of venous congestion leading to hyperexcitability of the neurones. The simplicity of this theory has made it attractive and it has been widely accepted, although explanations of the cerebral anæmia other than by compression, such as widespread tearing of the neuromuscular apparatus of the arterioles or excessive stimulation or paralysis of the vasomotor centre, have been offered.

According to Ricker,[3] damage to the neuromuscular mechanisms of the cerebral blood vessels first of all causes excessive vascular dilatation with consequent slowing of the blood stream and then extreme constriction which leads to ischæmia In fatal cases of cerebral trauma widespread vascular changes in the brain may

[1] Trotter, W "Injuries of the Skull and Brain" Choyce, "System of Surgery," **3**, 358 Cassell & Co London, 1932 "An Address on the Evolution of the Surgery of Head Injuries" *Lancet*, 1930, **1**, 169 "An Address on the Management of Head Injuries" *Lancet*, 1925, **2**, 953

[2] Kocher, T *Nothnagel's specielle Pathologie u Therapie IX Wien*, 1901

[3] Ricker, G *Virchow's Arch f Path Anat u Physiol u für Klin Med*, 1919, **226**, 180

often be demonstrated histologically, and Winkelmann and Eckel [1] have shown that the capillaries as well as the venules are congested, which suggests that the vascular changes are the result of active participation of the vessel walls in this state rather than the consequence of pure mechanical obstruction due to increased intracranial pressure. Swelling of the endothelial cells is an early occurrence, and from this stage up to complete occlusion of the lumen of the vessel all degrees of endarteritic change have been described.

It will be seen, therefore, that though microscopical evidence cannot prove that cerebral anæmia is responsible for the production of the phenomena of concussion, it does show that diffuse vascular changes *are* present in the brain, and these presumably are important. Certainly it is stasis of the blood flow which accounts for small foci of softening and for those areas from which the neurones so strangely disappear.

Theory of Physiological Neuronal Injuries.—For many hundreds of years it has been known that concussion may be caused by a slight shake up of the head which does not produce a demonstrable cerebral injury and is unassociated with signs of violence, either in the skull or in the overlying integuments.

In 1705 Littre [2] published a case which stimulated widespread interest in the pathology of concussion, and it is from this time that the conception of this state took on some kind of definite form. Littre's case was that of a strong young criminal who, wishing to forestall his sentence of the rack, took his measure of the cell where he was confined and, crouching with hands behind his back, ran towards the opposite wall, hitting his head against it with all his strength. He fell dead on the spot without saying a word or uttering a single cry.

M. Littre, called to inspect the corpse, began by examining the outside of the head. He was surprised to find there was no contusion, open wound or fracture. He then cut and separated all the integuments of the skull at the top of the head where, according to the evidence of several other criminals who had witnessed the act, the impact had occurred. He examined these integuments also from inside and found there no more alteration than had been found on the outside. He did not notice any injury to the skull bones after having uncovered these, except that the scaly part of the right temporal bone was separated from the parietal for a short distance.

It was therefore necessary for him to saw the skull and examine

[1] Winkelmann, N W, and Eckel, J L "Brain Trauma Histopathology during the Early Stages" *Arch Neur and Psych*, 1934, **31**, 956

[2] Littre "Diverses Observations Anatomiques" *Hist de l'Acad Roy des Sciences*, 1705, 54

the brain. This he did, but M. Littre's surprise grew when he found everything in a quite normal state and perfectly healthy. The only unusual thing was that the brain did not nearly fill the whole interior capacity of the skull, and its substance, as well as that of the cerebellum and spinal cord, was firmer and more solid to the sight and touch than normal. M. Littre made more certain of the obvious cerebral shrinking by putting the cut pieces of brain and the skull back in their place, which he was able to do very easily, although in other cases this could only be done with great difficulty.

His conclusion, therefore, was that the brain had sunk in very considerably with the violent disturbance of the blow, and as it had little elasticity it was not able to spring back to its former position. Consequently the distribution of nervous impulses in the remainder of the body, essential for all movements, had ceased at once.

As the report of this case was so important it was thought advisable to reproduce it in the above manner rather than to paraphrase it.

In 1866 Erichsen[1] came to the conclusion that spinal concussion was due to a molecular disarrangement of the cells concerned, and this probably led to the theory of cerebral concussion known as commotio cerebri or commotion of the brain cells.

To-day it is a firmly accepted belief that the brain can receive a purely non-structural physiological injury sufficient to produce a diffuse neuronal paralysis. This belief, of course, is of paramount importance, as it must influence profoundly any attitude in regard to treatment of concussion, since anything mechanistic such as decompression is unlikely to improve this state. The evidence in support of the physiological theory is largely forthcoming from experimentation in which the brains of animals whose heads have been subjected to small repeated blows sufficient to cause concussion have shown negative macroscopical or microscopical findings.[2]

Recently Brown and Russell[3] have proved by experiment that concussion may be caused by the forces of acceleration alone, that the resulting neurological state is of the non-structural type and that unconsciousness is probably due principally to injury of the medulla.

The Theory of Organic Neuronal Injury.—The essential principle of this theory is that unconsciousness and allied

[1] Erichsen, J E "On Concussion of the Spine, Nervous Shock and other Obscure Injuries to the Nervous System" New Edition Wm Wood & Co Baltimore, 1886

[2] Jakob, A *Nissl-Alzheimer's Histologie u histopath Arbeiten uber die Grosshirnrinde Jena*, 1912, **5**, 182

[3] Denny-Brown, D E, and Russell, W R "Experimental Concussion" *Proc. Roy Soc Med*, 1941, **34**, 691

neurological states can be explained by demonstrable changes in the neurones themselves, which means that concussion is based on morbid anatomy. From the nature of any severe accident in man it is reasonable to assume that diffuse and organic neuronal changes must be frequent, since the head is not subjected to small repeated blows used in the production of concussion experimentally but receives a single severe violence sufficient to deform the whole brain, and deformity implies bending, stretching or tearing. Histologically, neuronal changes distant from the sites of contusion have been proved by the studies of Rand and Courville,[1] and Greenfield,[2] though the latter writer believes that they might be secondary to œdema. In my opinion, demonstrable changes in neurones other than in contusional areas can always be found following fatal injuries, and particularly if the patient has survived for more than twenty-four hours These findings suggest that concussion may be due to organic neuronal injury, but nothing more definite can be claimed for them since correlation of loss of function with anatomical change is always conjectural. Furthermore the distribution and exact number of injured neurones necessary for the production of the phenomena of concussion are not known, and therefore at this stage it would be opportune to consider which part of the brain is concerned with consciousness and what is meant by unconsciousness. Although unconsciousness is often appreciable at a glance its definition is extremely difficult, and possibly new words will have to be coined before a completely satisfactory one is produced. In this state there is a cessation of those mental processes which normally can be engaged by an observer. Whether the patient is totally unable to think even in semicoma is not known, but judging from restlessness some form of intellectual activity must remain, otherwise his movements can only be regarded as a form of epilepsy of the postural centres Moreover a semiconscious patient, driven by the discomfort of a distended bladder, may attempt to get out of bed to relieve himself, and this is indicative of a complex mental process rather than of a pure reflex phenomenon. Reactions to external environment are changed, and in particular an unconscious patient is unresponsive to stimulation of sight and hearing. Muscle power and co-ordination may be largely retained, but the patient seems unwilling to use his limbs except for purposes of defence These various points are embraced in Mapother's [3] definition of con-

[1] Rand, C W, and Courville, C B "Histologic Studies of the Brain in Cases of Fatal Injury to the Head (5) Changes in the Nerve Fibres" *Arch Neur and Psych*, 1934, **31**, 527-555

[2] Greenfield, J G "Some Observations on Cerebral Injuries" *Proc Roy Soc Med*, 1938, **32**, 43 52

[3] Mapother, E "Mental Symptoms associated with Head Injury The Psychiatric Aspect" *Brit Med Jour*, 1937, **2**, 1055-1061

follow soon after the receipt of an injury and which is not due to hæmorrhage. It commonly occurs in acute cerebral trauma and usually responds rapidly to warmth, rest and relief of pain. Its underlying physiopathological cause is not known. Probably in cerebral trauma it is due to vasomotor paralysis of central origin and is part of the phenomena of concussion.

Secondary shock is a condition of circulatory failure. It develops insidiously some hours after injury and may be induced by cold, pain, hæmorrhage or toxins. Exposure in road accidents or following air raids is not very common, as the injured are so rapidly cared for and transferred to hospital. It is on the battlefield and on the mountains where the factor of cold is important The combination of chilling and severe concussion is almost invariably fatal In climbing accidents there would be fewer fatalities if the patient were thoroughly warmed in a small tent or by any other means before an attempt were made to carry him down the mountainside. Pain and discomfort are common in the early stages of acute cerebral trauma. Often they are due to an irritative meningitis consequent on a subarachnoid hæmorrhage. Pain not only causes shock directly but also induces restlessness leading to exhaustion and to aggravation of any condition of collapse. Loss of blood in so-called "closed injuries" is often considerable, and even small hæmorrhages may produce adverse effects in those cases where the cerebral circulation is already embarrassed by other conditions. Profuse bleeding may occur from the nose, ears, cuts on the scalp, into the subgaleal and subarachnoid spaces or from wounds elsewhere in the body. Severe bruising of the face and crushing wounds of the limbs and body are often associated with states of concussion, and if it is agreed that absorption of broken-down tissue protein can cause secondary shock then toxæmia may often be responsible for this state in acute head injuries.

Whatever may be the precipitating factors of secondary shock the essential cause is diminished blood volume, with a consequent fall in heart output and blood-flow through the tissues of the body. The loss of blood volume in hæmorrhage is obvious. In other cases it is due to loss of plasma into the tissue spaces of the body, and this is dependent on stasis of capillary circulation and on abnormal permeability of the capillary walls. As the arterioles are contracted some kind of vasomotor tone must be present, and thus the underlying sequence of events which leads to secondary shock in non-hæmorrhagic cases commences at the stage of capillary paralysis

In a conscious patient secondary shock is characterised by weakness, pallor, a rising pulse rate, a falling blood pressure,

impulses are initiated, or at their axons along which the impulses are conducted, and thus widely separated lesions can give rise to the same neurological signs.

In my opinion traumatic unconsciousness is usually due to paralysis of the cortical cells or to blockage of the subcortical pathways, and only occasionally to local injury within the basal ganglia or brain stem. Moreover I believe that there is no basic or uniform histological picture on which variegated contusional lesions may be superimposed that will account for the phenomena of concussion. A so-called physiological injury is probably but a minor degree of the kind in which structural change can be demonstrated, and presumably both physiological and organic neuronal injuries occur in different parts of the same injured brain. In the cases where physiological injury is the dominant feature, recovery is probably rapid, whereas it is relatively slow when an organic injury predominates. In both types depth of unconsciousness is dependent on the number of neurones paralysed. A force sufficient to interfere with the vascular mechanism of the brain is unlikely to leave intact the more delicate neurones, and on evidence so far available it is difficult to believe that traumatic unconsciousness is ever produced by circulatory changes alone. On the other hand, circulatory disturbances often intensify the effects of direct neuronal injury whether of the organic or physiological type and may render permanent what otherwise may have been a reversible injury. Physiological injuries no doubt will be proved to have a structural basis when three-dimensional views can be made on brains not subjected to processes (hardening, dehydration, staining and cutting) which inflict more damage on the tissues than the injury itself.

My belief is that in man prolonged concussion following severe violence to the head is due to direct diffusely scattered injury of an organic type to a large number of neurones.

SECONDARY PATHOLOGICAL MANIFESTATIONS

The degree and extent of a primary injury is determined at the moment of impact, and if that part of the brain which is essential to life has not been destroyed by contusion, laceration or diffuse neuronal injury, the natural tendency of the patient will be towards recovery if other factors do not supervene Unfortunately secondary developments are invariable and often render fatal what otherwise would have been a reversible injury.

Shock.—Primary shock, according to a report issued by the Medical Research Council,[1] is a condition of collapse which may

[1] Medical Research Council "The Treatment of Wound Shock" M R C War Memorandum No 1 H M Stationery Office London, 1940

limited by an adherent dura. Intrasinus pressure, normally low, is greatly increased by the slightest obstruction to respiration or by compression of the veins of the neck by tight clothing or bandaging. Any increase in venous pressure results in commensurate increase in bleeding.

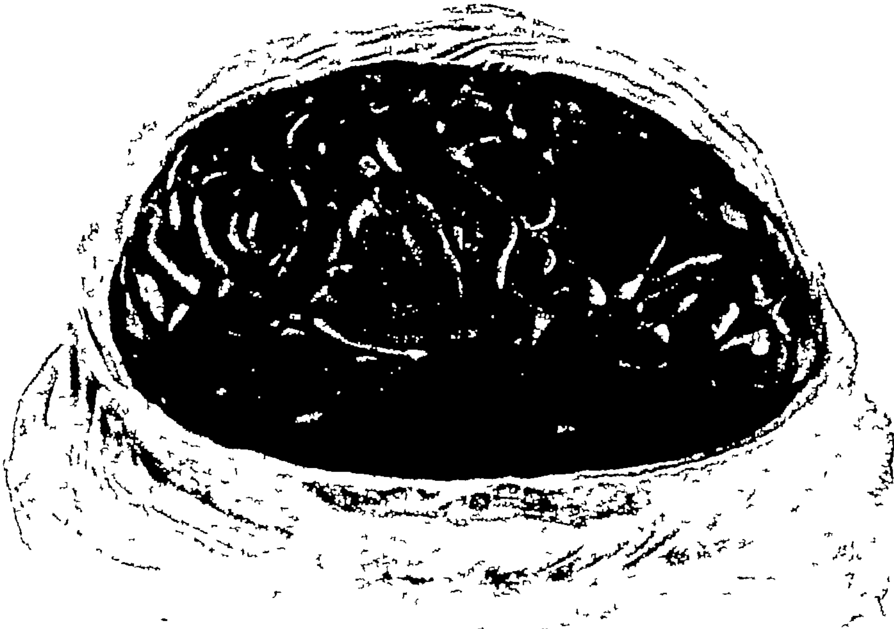


FIG 24

An extradural clot due to rupture of the middle meningeal vessels complicated by a subarachnoid hæmorrhage Such complications often explain why operations on extradural hæmorrhages are occasionally unsuccessful

Usually a tear takes the form of a small triangular flap and less commonly of a transverse or longitudinal split. Complete transections occasionally happen in open wounds, but in closed head injuries they are almost unknown and in both cases are invariably fatal.

In view of the frequency with which basal fractures converge on the pituitary fossa, injuries of the cavernous sinus are surprisingly rare, the superior longitudinal and lateral sinuses being those most commonly affected.

Diploic Bleeding.—Probably diploic bleeding is the most frequent cause of extradural hæmorrhage. Often not more than a thin layer of blood collects; as this does not cause symptoms of cerebral compression it passes unrecognised and is absorbed by natural processes. Profuse diploic bleeding usually comes from numerous and widespread points, the precise localisation

sweating, vomiting and intense thirst Such a picture, of course, in an unconscious patient is very difficult to distinguish from that due to a failing cerebral circulation directly consequent on an injury to the brain. In other words, secondary shock and a deteriorating state of concussion are almost indistinguishable.

In a certain and important group of injuries, which will be described in further detail later, a patient may remain in a state of semicoma for over twenty-four hours without the depth of unconsciousness or the general neurological picture altering one way or another. The first sign of retrogression in such cases may be an increase in pulse rate and a fall of diastolic blood pressure. Some of these adverse changes are due to secondary shock, and lives may be saved by an early blood transfusion.

As regards prognosis, a reduction of the blood volume by 25 per cent produces few symptoms, whereas a further reduction of 25 per cent. is serious. A pulse rate below 110 and a systolic pressure above 95 mm. Hg. associated with a blood volume of 75 per cent. are good signs, but figures worse than these are evidence of very grave deterioration.

MASSIVE HÆMORRHAGES

Extradural Hæmorrhages.—Extradural hæmorrhages large enough to be of surgical significance are extremely rare and are found in about 1 or at most 2 per cent. of cases in any large series of acute cerebral trauma.

They are, of course, extremely important, and it must be realised that the bleeding may come not only from the middle meningeal vessels but also from the diploic veins or dural venous sinuses (Fig. 24). Occasionally they may be associated with subarachnoid hæmorrhages or intrinsic damage to the brain.

Sinus Bleeding.—Tears in the walls of the dural venous sinuses are nearly always associated with overlying fractures Fortunately they are rare, and this is due no doubt to the fact that the large dural channels, with the exception of the descending limbs of the lateral sinuses, are not embedded in deep bony grooves but run in contact with the flat surfaces of the skull and so are not necessarily lacerated when the overlying bone is fractured As the sinus walls are fibrous and rigid, they do not collapse or contract when torn as arteries do, with the result that bleeding is often profuse in spite of low venous pressure, and may be so rapid that the extravasated blood has not time to clot before the patient succumbs Bleeding, however, is not invariably fatal, because the spicule of bone which lacerates the sinus may plug the opening into it, or, as occasionally happens, the hæmorrhage may be

recovers and the blood pressure rises, rapid and profuse bleeding may occur The latent interval which so commonly occurs in middle meningeal hæmorrhages is not always due to shock but often can be accounted for by the ability of the brain to accommodate itself to a slowly expanding lesion for a long time before showing signs of compression In many cases the vessel bleeds from the moment the injury is inflicted, but often at a slow rate, due partly to incomplete rupture and partly to the resistance of the dura to stripping. Immediate prodromal symptoms such as headache are the rule rather than the exception, and the rapid development of neurological signs later indicates loss of com-

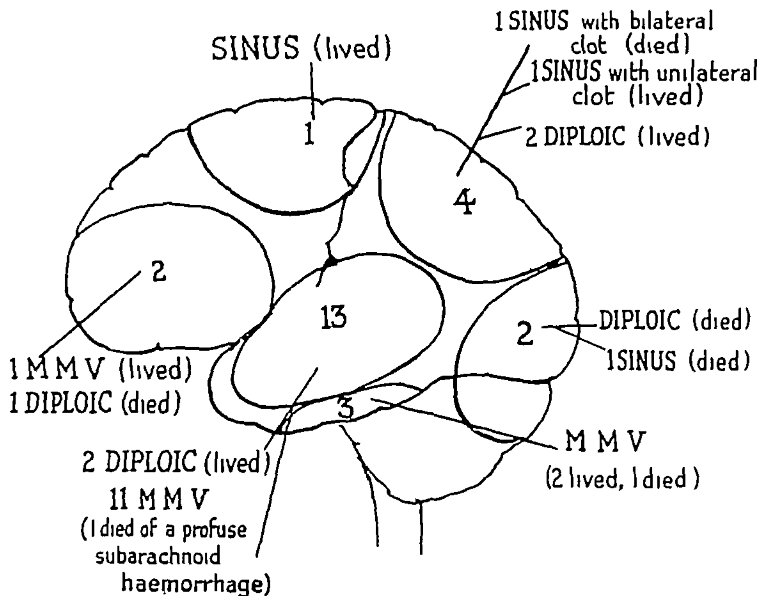


Fig 25

Analysis of cause and position of twenty-five cases of extradural hæmorrhage M M V, Middle meningeal vessels, Sinus, tear of dural venous sinus, Diploic, bleeding from fractured bone

pensation in the brain rather than a sudden severe hæmorrhage. Clots usually take the form of a segment of a sphere, the summit of which corresponds with the bleeding point on the dura, and they may be found in any position within the skull. But as will be seen below, they are most commonly found over the outer surface of the temporal lobe

Analysis of Twenty-five Cases of Extradural Hæmorrhage.— In 25 cases of extradural hæmorrhage on which I have operated, there was a latent interval in every case varying from two hours to seven days, the usual period being round about four hours before the first serious sign developed The ages of the patients ranged from nine to sixty years. Thirteen hæmorrhages were found over the outer surface of the temporal lobe: 3 were basal, 2 were low in the frontal area; 1 was high over the

of which is impossible on clinical grounds since neurological signs point to the position of the clot or accumulation of blood and not to the origin of the bleeding which may be, and often is, some distance away. Radiology certainly shows the position and length of fracture lines from which the bleeding may emanate, but as these are often multiple and extensive, X-rays can rarely give information of more than lateralising value. The main difficulty in treatment is that the bleeding points may not be accessible through the exposure necessary for the evacuation of the clot or pool of blood which is compressing the brain.

Middle Meningeal Hæmorrhages.[1] [2]—The middle meningeal artery arises from the internal maxillary branch of the external carotid artery, and enters the middle fossa through the foramen spinosum which lies just behind and lateral to the third division of the trigeminal nerve. Throughout its intracranial course it is accompanied by two venæ comites, which means that whenever the meningeal vessels are ruptured, bleeding takes place from both ends.[3] From the foramen spinosum the artery runs forwards and outwards on the base of the skull towards the tip of the great wing of the sphenoid bone, where it divides into an anterior and posterior branch. The anterior branch continues upwards and forwards to the antero-inferior angle of the parietal bone, which it deeply grooves or tunnels, and then turns upwards and backwards towards the vertex. The posterior branch runs backwards and upwards across the squama of the temporal bone to the occipital region, this being the branch most commonly exposed in temporal decompressions.

A point of interest is that the grooves in the bone caused by the anterior branches are commonly visible in X-rays and may be confused with fracture lines. Apart from its named branches, which are of little surgical importance, numerous small twigs run into the bone and are very easily avulsed whenever the dura moves away from the bone as the skull is deformed Inosculations across the middle lines between the vessels of either side are not very free, but they are widespread between the anterior and posterior branches on each side.

The vessels may be ruptured in many ways. They may be transfixed by a spicule of bone or lacerated by the edge of a fracture or torn by stretching, as described in the previous chapter. Bleeding may not take place immediately when the vessels are torn, owing to the influences of shock, but as the circulation

[1] M'Kenzie, K G "Extradural Hæmorrhage" *Brit Jour Surg*, 1938, **26**, 346
[2] Jacobsen, W H A "Middle Meningeal Hæmorrhage" *Guy's Hospital Report*, 1885-86, **43**, 147
[3] Wood Jones, F "The Vascular Lesion in Some Cases of Middle Meningeal Hæmorrhage" *Lancet*, July 1912, **1**, 7

Acute Subdural Hygromata.[1 2]—Hygromata may be due to laceration of the arachnoid which allows cerebrospinal fluid to escape into the subdural space. When large collections of fluid accumulate they are probably due to the tear in the arachnoid acting as a one-way valve, which allows fluid into the subdural space but not out of it. An alternative theory is that subdural hygromata are the result of transudations into the subdural cavity due to injury of the lining cells, and this theory is supported by the fact that the protein content of the fluid is often much higher than that of the cerebrospinal fluid.

Chronic Subdural Hæmatomata.—Chronic subdural hæmatomata have aroused widespread interest not because of their frequency, which in fact is less than 1 per cent. in any clinical series of acute cerebral trauma, but rather to the problems of their development and, to a lesser extent, the excellent results which are obtained by drainage.

The condition "pachymeningitis hæmorrhagica interna" has been recognised at least since the middle of the last century and was fully described by Virchow[3] under the term "hæmatoma duræ matris." None the less, it remained for Trotter[4] to stress the hæmatomas' traumatic origin and to show that they often follow a very slight knock on the head.

The course of events in the development of any chronic subdural hæmatoma is complicated.[5 6] A vessel, probably a vein, ruptures and then seals and the resulting hæmatoma becomes enveloped in a fibrinous membrane which later is organised by mesothelial invasion. The part of the membrane nearest the arachnoid remains thin and non-adherent, whereas the outer part thickens and becomes attached to the dura and impossible to strip without rupturing numerous small blood vessels, which is a point of some surgical importance. At some phase in its life-history the encapsulated clot begins to swell, but why this happens is not known. It has been suggested that small repeated bleedings could account for it, and this view is supported by the fact that at autopsy cerebral veins have been found either attached to the capsule or thrombosed within the clot Putnam[7 8] showed

[1] Naffziger, H C "Subdural Fluid Accumulations following Head Injury" *J A M A*, 1924, **82**, 1751

[2] Hardman, J "Assymetry of the Skull in relation to Subdural Collections of Fluid" *Brit Jour Radiology*, 1939, **12**, 455-461

[3] Virchow, R *Verhandl d phys-med Gesellsch*, 1857, **7**, 134

[4] Trotter, W "Chronic Subdural Hæmorrhage of Traumatic Origin and its relation to Pachymeningitis Hæmorrhagica Interna" *Brit Jour Surg*, 1914, **2**, 271-291

[5] Martin, J P "Chronic Subdural Hæmatoma" *Proc Roy Soc Med*, 1930-31, **24**, 585-590

[6] Miller, C R "Chronic Subdural Hæmorrhage" *Med Bull Veterans' Adm*, **11**, 332-336 Washington, 1934-35

[7] Putnam, T J, and Cushing, H "Chronic Subdural Hæmatoma" *Arch Surg*, 1925, **11**, 329

[8] Putnam, T J, and Putnam, I K "Experimental Study of Pachymeningitis Hæmorrhagica" *Jour Nerv and Ment Dis*, 1927, **65**, 260

front part of the motor cortex; 4 were high in the parietal area and 2 were low over the occipital cortex, 1 of which extended into the posterior fossa. In 15 cases the middle meningeal vessels were ruptured; in 6 cases the bleeding came from the diploic veins and in 4 cases from tears in the walls of the dural sinuses. Nineteen out of the 25 patients operated on lived and 6 succumbed. Of the deaths, 2 were the result of middle meningeal hæmorrhage, 1 of which was complicated by a profuse subarachnoid hæmorrhage (Fig. 25); 2 were the result of diploic bleeding and 2 the result of sinus bleeding.

Subdural Hæmorrhages.—The arachnoid and dural membranes normally lying in close contact with each other are readily separable

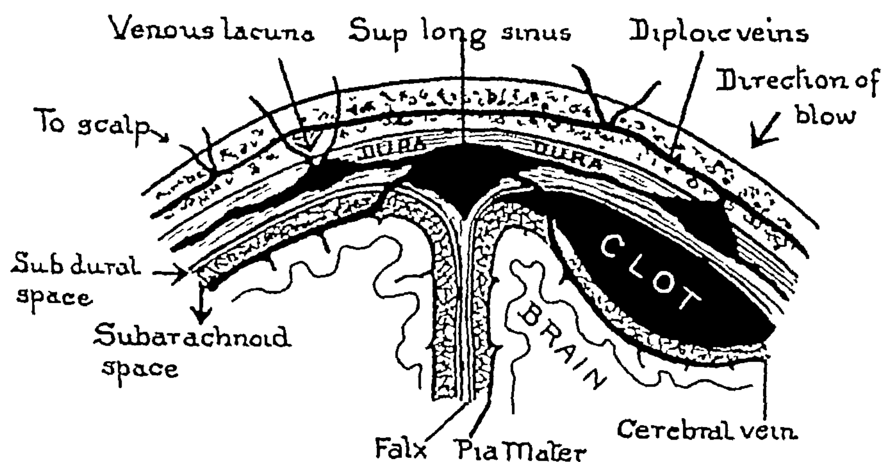


Fig 26

The tributaries of the parasaggital sinus The communicating veins which run from the cortical vessels into the sinus are commonly ruptured when the brain moves across the face of the dura In this case a unilateral subdural hæmatoma is shown

and enclose a potential space which can be converted into an extensive cavity by a hæmorrhage or leakage of cerebrospinal fluid into it.

Acute Subdural Hæmatomata.[1]—Bleeding into the subdural space may come from a dural venous sinus or a cortical vein if the arachnoid has been torn or, as is most common, from the short communicating veins which drain the cortical vessels into the sinuses (Fig. 26). Usually the bleeding is slight, but occasionally is profuse and may lead to fatal compression within a few hours Often it is bilateral, and the blood tends to trickle to the most dependent part of the subdural space according to the position of the head, and is usually found in greatest bulk over the outer surfaces of the temporal lobes.

[1] Munro, D "Craniocerebral Injuries, their Diagnosis and Treatment" Oxford, 1938.

(ii) Absence of coagulation.
(iii) Pink, brown or yellow colouration of the clear supernatant fluid when the red cells have been allowed to sink to the bottom of the tube.

Extravasated blood is rapidly hæmolysed and the products removed via the arachnoid villi, but xanthochromic staining of the cerebrospinal fluid may persist for many weeks after all active bleeding has ceased.[1] The results of subarachnoid bleeding, when superimposed on the effects of the primary injury, are extremely serious. The irritation of the blood in the meninges, for instance, causes restlessness, with resulting cerebral congestion. This may cause increased bleeding or precipitate cerebral œdema. Blood loss may lead to a measurable degree of anæmia, and large quantities of extravasated blood have the compressive effect of a large clot. Also, resulting meningeal adhesions lead to faulty circulation or absorption of the cerebrospinal fluid.

The amount of blood in the cerebrospinal fluid is of prognostic significance only within very wide limits, and more will be said on this subject in the following chapter.

Subpial Bleeding.—The pia mater is firmly attached to the cortex of the brain and cannot be freely stripped, so that a subpial hæmorrhage always appears as a stain on the surface and never as a thick clot, although it may be so diffuse that it virtually constitutes a massive hæmorrhage. Usually it is an integral part of the contusion.

Intracerebral Bleeding.[2, 3]—Large hæmorrhages deep in the tissues of the cerebrum, cerebellum or brain stem occasionally occur, and usually are due to pre-existing disease in the cerebral vessels On the other hand, large clots near the surface, the result of lacerations, are by no means uncommon.[4]

Intraventricular Hæmorrhages. — Profuse intraventricular hæmorrhages are commonest in children They are invariably fatal and are due either to tearing of the choroid plexus or to rupture of a large intracerebral hæmorrhage through the ventricular walls [5]

Œdema.—The term "cerebral œdema" implies that the brain is swollen, owing to an increase of fluid in the pericellular and

[1] Bagley, C "Blood in Cerebrospinal Fluid Experimental Data" *Arch Surg*, 1928, **17**, 18
[2] Craig, W McK, and Adson, A W "Spontaneous Intracerebral Hæmorrhage Etiology and Surgical Treatment with a Report on Nine Cases" *Arch Neur and Psych*, 1936, **35**, 701
[3] Martland, H S, and Beling, C C "Traumatic Cerebral Hæmorrhage" *Arch Neur and Psych*, 1929, **22**, 1001
[4] Friedman, E D "Massive Intracerebral Hæmorrhage of Traumatic Origin Injuries of the Skull, Brain and Spinal Cord" Baillière, Tindall & Cox London, 1940
[5] Hemsath, F A "Ventricular Cerebral Hæmorrhage in the New-born" *Amer Jour Obst and Gyn*, 1934, **28**, 343

that the adventitious membrane enveloping the clot becomes highly vascularised, and he suggested that oozings from the newly formed vessels would account for any increase in size of the hæmatoma. In favour of this theory is the common finding of fresh blood within the clot, even after a considerable period has elapsed since the injury. A popular and attractive theory is that of Gardner,[1] who proved that the membrane of the hæmatoma possesses semipermeable properties. He therefore offered the explanation that, as the clot disintegrates, its molecular concentration will increase and cause a rise in osmotic pressure, with the result that cerebrospinal fluid will be drawn through the semipermeable membrane by the processes of dialysis. In favour of this hypothesis are the varied contents which may be found in the hæmatoma. These may be partly solid and partly fluid, or they may be wholly fluid. Also the fluid may be black and viscous or it may be light brown and watery. Furthermore, Munro has shown that the protein content of the fluid obtained from these hæmatomata actually increases as they break down. On those occasions when the hæmatoma is not drained or removed by dissection it may fibrose or even calcify.[2]

Subarachnoid Bleeding.—Bleeding into the subarachnoid space is the most important and by far the most common form of massive bleeding due to acute cerebral trauma. In those patients who have been unconscious for over one hour, it is found in at least 75 per cent. of cases, and according to Nielson and Rand[3] the figure is as high as 85 per cent The bleeding may come from any vessel on any surface of the brain. In profuse hæmorrhages the communicating veins draining into the dural sinuses have usually been torn The blood collects chiefly in the sulci, which it appears to fill, and also mixes freely with the cerebrospinal fluid, which prevents its clotting. Thus it is carried to all parts of the cerebral and spinal subarachnoid spaces and cisterns Extensive intracranial clotting may occur when a large vessel is severed, but such occurrences are usually rapidly fatal.

According to Symonds,[4] the distinctive features of the cerebrospinal fluid taken at lumbar puncture are :—

(1) An even admixture of the blood which is the same in a series of specimens collected at the same puncture.

[1] Gardner, W J "Traumatic Subdural Hæmatoma, with particular reference to the Latent Interval" *Arch Neur and Psych*, 1932, **27**, 847-858

[2] Schüller, A "Hæmatoma duræ matris ossificans" *Fortschr a d Geb d Rœntgenstrahlen*, 1935, **51**, 119

[3] Nielson, J M, and Rand, C W "Fracture of the Skull Analysis of One Hundred and Seventy-one Proved Cases Diagnosis and Treatment of Associated Brain Injury" *Arch Surg*, 1925, **11**, 434 458

[4] Symonds, C P "Spontaneous Subarachnoid Hæmorrhage" *Proc Roy Soc Med*, 1924, **17**, 39 52

been reached on this point, but in neurological and neuro-surgical circles it is believed that generalised œdema, as distinct from local œdema around a contusion, occurs much less frequently than has hitherto been supposed.[1] My own opinion on the incidence of œdema is that the generalised form is rare, whereas local swelling around a contusion is invariable. Furthermore, no one yet has been able to prove satisfactorily what part œdema plays in the production of unconsciousness or other neural dysfunction, and I believe it is not nearly so important a factor as subarachnoid hæmorrhage.

That generalised œdema can occur there is no doubt. When operating under local anæsthesia, I have occasionally found the brain, in the acute phases of a head injury, under such great tension that it bulged into the wound as soon as the dura was opened, and later at autopsy have been able to show that the increased tension was not due to hydrocephalus and could not have been due to the amount of blood extravasated. It cannot be objected that the increased tension found on surgical exploration was due to the anæsthetic because local anæsthesia does not affect intracranial tension.

The accumulation of the excess fluid in cerebral œdema probably is due to increased capillary permeability consequent either upon abnormal metabolites in the interstitial spaces, or on vasomotor paralysis in the region of the brain concerned. When the condition of œdema has developed, venous congestion will tend to perpetuate it. In fact, a vicious circle results. Œdema leads to increased intracranial tension, this causes a venous congestion, and venous congestion, by causing further capillary stasis, increases the œdema

Hydrocephalus.—Hydrocephalus is a state of dilatation of the cerebrospinal fluid spaces, and there are two main varieties—internal and external.

Internal hydrocephalus implies that the ventricular system only is affected. It is always caused by an obstruction, and this may be situated at the foramen or foramina of Monro, in the Sylvian aqueduct or at the foramina of Majendie and Luschka. According to the site of the obstruction, the ventricular system proximal to the block becomes dilated and thus one or all the ventricles may be involved

As a complication in the acute stages of a cerebral injury internal hydrocephalus is rare, and when it does occur it is usually due to occlusion of the Sylvian aqueduct by a clot of blood seeping downwards from a lateral ventricular hæmorrhage. As a late sequel it is also rare, and in this case may be due to (1) stricture

[1] Greenfield, J G Personal communication

perivascular spaces. It does not refer to the amount of cerebrospinal fluid in the ventricles or in the subarachnoid spaces, although it is probable that all the reservoirs of the brain act dependently. Also, it must be distinguished from "swelling of the brain," which is thought to be due to hydration of the cells themselves. Histologically, the picture of cerebral œdema is very typical; the tissues appear areolar or honeycombed, owing to the distension of the spaces mentioned above (Fig. 27). Œdema may develop early or late, and may be transient or persistent. It may affect



FIG 27
The honeycombed appearance, typical of cerebral œdema

the whole brain or be confined to a lobe or, as is more usual, be localised around an area of contusion.

Since the condition of œdema profoundly influences the mode of treatment in the acute stages of a closed head injury, it is important to inquire whether it occurs as often as is so commonly supposed and to what extent it may embarrass the cerebral circulation or interfere with the nutrition of the neurones. In America the consensus of opinion is that œdema of a serious degree almost invariably accompanies a severe injury of the brain and often causes a rise in intracranial pressure with serious consequences [1-3] In this country no definite conclusion has yet

[1] Fay, T "The Treatment of Acute and Chronic Cases of Cerebral Trauma by Methods of Dehydration" *Ann Surg*, 1935, **101**, 76-132

[2] Lecount, E R, and Apfelbach, C W "Pathologic Anatomy of Traumatic Fractures of Cranial Bones and Concomitant Brain Injuries" *J A M A*, 1920, 501

[3] Rand, C W "Histologic Studies of the Brain in Cases of Fatal Injury to the Head Preliminary Report" *Arch Surg*, 1931, **22**, 738-753

findings in one case at post mortem. These were as follows: the left basal cisterns were blocked with a large clot of blood and the cortex of the brain on this side was firmly pressed against the dura, whereas on the right side the basal cisterns were free and the cortex covered by a deep external hydrocephalus. Therefore, as there were no signs of bleeding on the left side, and as the arachnoid villi could not have been plugged with blood cells, it may be assumed that the obliteration of the cerebrospinal

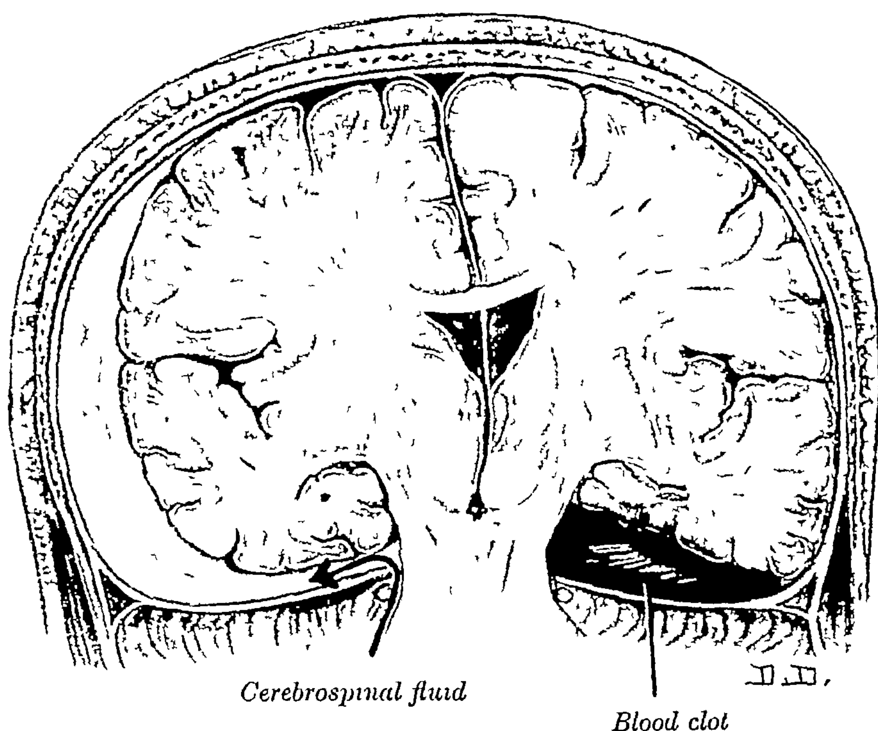


FIG 28
An external hydrocephalus, the result of a traumatic basal hæmorrhage

fluid spaces on the left side led to the hydrocephalus on the right side of the brain (Fig. 28).

Herniations of the Brain.—Neurological signs in head injuries are produced in a diversity of ways. They may be caused by (1) direct injury to the neurones; (2) by anæmia, local and general; (3) by circulatory anomalies of the cerebrospinal fluid; (4) by the strangulation of structures, over dural edges or taut vessels; or (5) by foraminal herniations. It is with foraminal herniations that we are particularly concerned in this section.

Whenever the brain swells or whenever the intracranial space is encroached upon by an expanding lesion, such as an extradural hæmorrhage, cerebrospinal fluid is forced into the spinal theca.

of the Sylvian aqueduct consequent on bruising of its walls, (2) adhesions in the posterior fossa or (3) traumatic cysts compressing the fourth ventricle.

External, or communicating, hydrocephalus implies that cerebrospinal fluid can escape from the ventricles on to the surface of the brain. It may be produced by (1) excessive secretion, (2) by faulty absorption, (3) by obstruction to the circulation of the cerebrospinal fluid or, as so often happens, by a combination of these factors. Although it is impossible to state in percentages the incidence of external hydrocephalus, there is much clinical and post-mortem evidence to show that it is a happening of considerable frequency and importance in the acute phases of a cerebral injury. In decompressions performed under local anæsthesia or when an inspection hole has been cut to establish a diagnosis, the bruised or normal cortex of the brain is often seen pushed away from the dura and not pressed tightly against it. Also, on making a small incision through the dura and arachnoid, a stream of yellowish or lightly bloodstained fluid will often spurt out under great pressure to a height of several inches.

In three cases of delayed hemiplegia—one examined at autopsy and two at surgical exploration—I have been able to prove beyond all doubt that the underlying pathological cause of the paralysis was an extensive unilateral external hydrocephalus.

The cause of external hydrocephalus is usually assumed to be faulty absorption of cerebrospinal fluid owing to blockage of the arachnoid villi by extravasated red blood corpuscles, and this explanation seems reasonable, since certain cells of the villous tufts are known to resemble those of the reticulo-endothelial system in their capacity to engulf and destroy foreign bodies.

Alternatively, Rand and Courville,[1] basing their views on a series of sixty-one autopsy specimens in which they demonstrated all degrees of injury to the epithelium of the choroid plexuses, concluded that such trauma would lead to excessive secretion of cerebrospinal fluid and to traumatic hydrocephalus.

Although blocking of the arachnoid villi and increased secretion of cerebrospinal fluid probably are the two main causes of any accumulation of excess fluid under tension in the subarachnoid spaces, interference with the circulation of the fluid is a causative factor which also must be taken into account, particularly since blood acts as a strong irritant to the leptomeninges and rapidly produces adhesions. That surface anomalies of cerebrospinal fluid circulation can produce hydrocephalus was proved by my

[1] Rand, C W, and Courville, C B "Histologic Studies of the Brain in Cases of Fatal Injury to the Head (2) Changes in the Choroid Plexus and Ependyma" *Arch Surg*, 1931, **23**, 357-425

cranial cavity, processes of brain tissue are apt to be forced through the openings of the hiatus tentorii or of the foramen magnum as elongated herniations.

The Tentorial Pressure Cone.—The tentorial pressure cone is a most important complication, and although it has been known for some time, it was so named by Jefferson,[1] who not only described its mechanics but illustrated its surgical significance.

During some phase in the rise of supratentorial pressure a process of brain tissue from the under and inner surface of the temporal lobe (uncus) herniates through the opening of the tentorium. The midbrain is displaced and compressed against the opposite free edge of the tentorium or, if the herniation is bilateral, between the two herniated processes. Conduction of impulses from the cerebrum is impaired, and the neural mechanisms below the compression are released from the control of influences of the higher centres. A primitive postural state known as decerebrate rigidity develops which is well known from animal experiment and follows transection of the brain stem just below the red nucleus. The muscles of the limbs go into a state of extreme hypertonus, usually in a position of extension but occasionally in flexion.

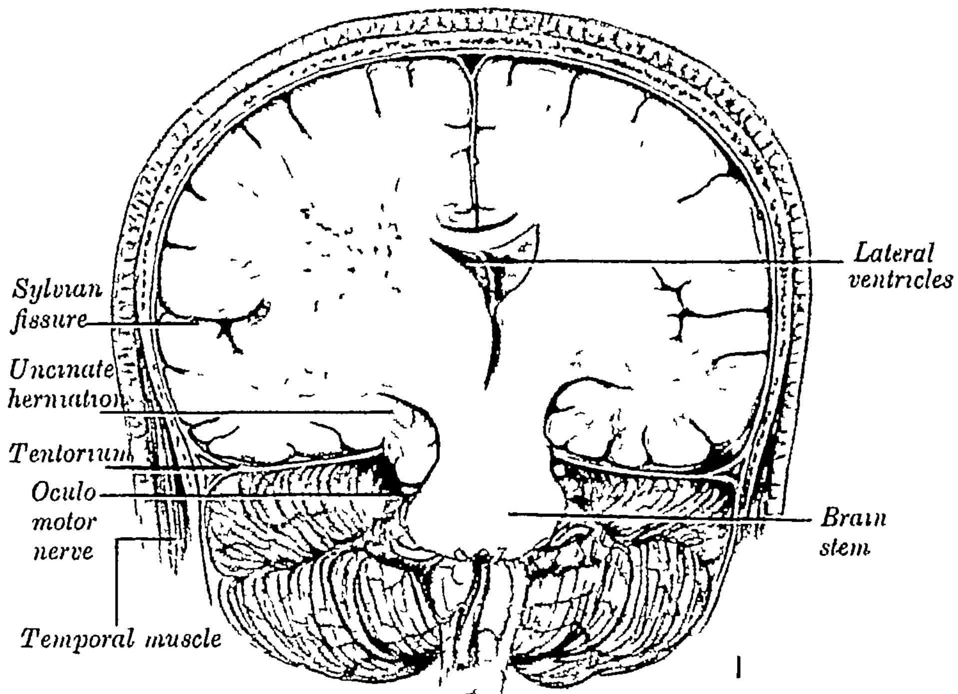
Another important complication is that the third nerve may be stretched or compressed by the herniation. It is this mechanism that accounts for a most important diagnostic sign, viz., the fixed dilated pupil. If not relieved, a tentorial compression soon leads to death in coma (Fig 29).

The Cerebellar Pressure Cone of Cushing.—A rise of pressure in the posterior fossa may force the tonsils of the cerebellum through the foramen magnum into the spinal canal with resulting compression of the medulla oblongata Such compression leads to respiratory embarrassment which might go on to complete failure. Although the circulatory centres may continue to function for a time, sooner or later they also fail and the patient dies. Fits in which the patient goes into opisthotonos occasionally occur. Cerebellar herniation is less frequent than tentorial herniation because it is the cerebrum and not the cerebellum that usually bears the brunt of an injury.

Meningitis and Encephalitis.—Bacteria may enter the intracranial cavity through the nose or ears and produce meningitis or encephalitis.[2] Abscesses of the brain, in the surgical sense, do not occur in the acute phases of head injury for the obvious reason that a capsule has not time to form before the major issues

[1] Jefferson, G "Tentorial Pressure Cone" *Arch Neur and Psych*, 1938, **40**, 857-876
[2] Macewen, W "Pyogenic Diseases of the Brain and Spinal Cord," 333 James Maclehose & Sons Glasgow, 1893

Later, the basal cisterns are obliterated and the ventricles become flattened. If the forces of compression or of œdema continue to



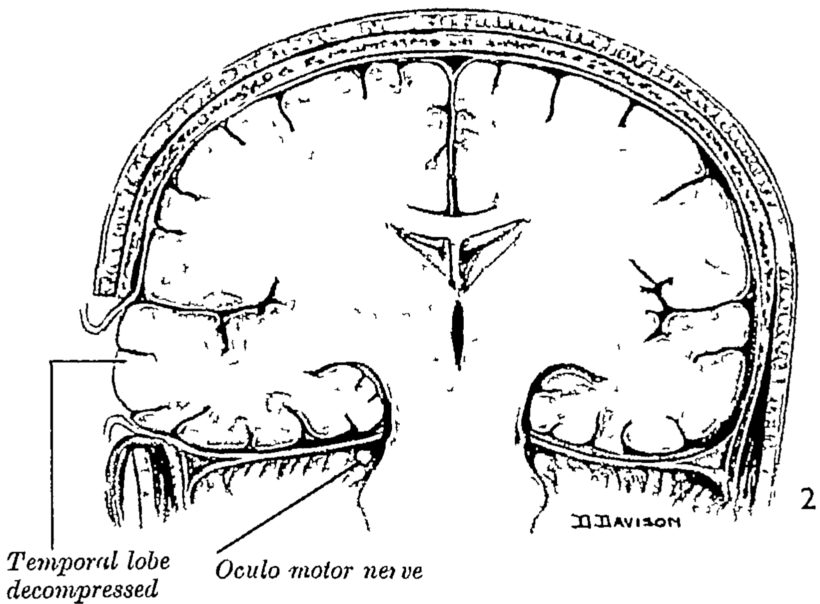


FIG 29
A tentorial pressure cone due to œdema, relieved by subtemporal decompression It is in this type of case that operative treatment gives the most spectacular results

act beyond the limits within which compensation is possible, by further expulsion of cerebrospinal fluid or of blood from the intra-

infections of those extravasations of blood which so commonly occur at the top of the nose, either in the extradural space or in the sinuses themselves.

In petromastoid injuries delayed infection is more frequent than immediate meningitis, and is due almost invariably to suppurative otitis media which may or may not have been present before the injury. Naturally both immediate and delayed infective complications, whether from the nose or ears, are predisposed to by pre-existing disease in the air sinus fractured.

Death in acute cerebral infections is due chiefly to malignant œdema (Fig. 30) consequent on spreading encephalitis and not on the toxæmia of meningitis or blockage of the cerebrospinal fluid pathways by meningeal adhesions.

DEVELOPMENT OF THE NEUROLOGICAL PICTURE AND CAUSE OF DEATH

The neurological picture immediately following any violence to the head is due to one or to a combination of the three primary types of injury. On this background further symptoms and signs are often rapidly added by secondary pathological developments. Traumatic pathological states are essentially complex, and injuries to the same part of the brain may give rise to totally different signs according to whether they excite or paralyse the cells concerned Complete loss of function in the motor cortex, for example, with paralysis of the corresponding limbs, will in itself cause no further change in the intracranial pathological state. On the other hand, hyperexcitability resulting in convulsive seizures will so raise venous, and thereby intracranial, pressure that further damage may be inflicted on the brain. This is also true of restlessness and delirium. The effects of straining on the volume of the brain and on the cerebral circulation have to be seen only once, when a bone flap is turned, for their importance to be appreciated The brain bulges ominously; vessels become engorged and large veins may rupture and lead to serious hæmorrhage.

The results of contusion or laceration, apart from the secondary developments, depend on the part of the brain affected. It is the brain stem and basal ganglia that are essential to life. So long as the brain stem continues to function, respiration and circulation adequate for life will be maintained. Hæmorrhages into the hypothalamus may destroy the controlling stations of the vegetative or autonomic system and cause metabolic crises, hyperthermia or acute perforation of the gastro-intestinal canal, any one of which may be fatal. Contusions of the cerebral and cerebellar

are settled. As compound fractures of the frontal bone are more common than compound petromastoid injuries, infections through the nose are more common than those through the ears. Meningitis and encephalitis are often due to mixed infections; streptococci, staphylococci, pneumococci and heterogeneous bacilli being the bacteria usually concerned.

Infection may come on within a few hours of an accident and run a very rapid course. This occurrence is often not so unfortunate as would at first sight appear, since it is usually associated with a severe contusion or laceration of the brain,

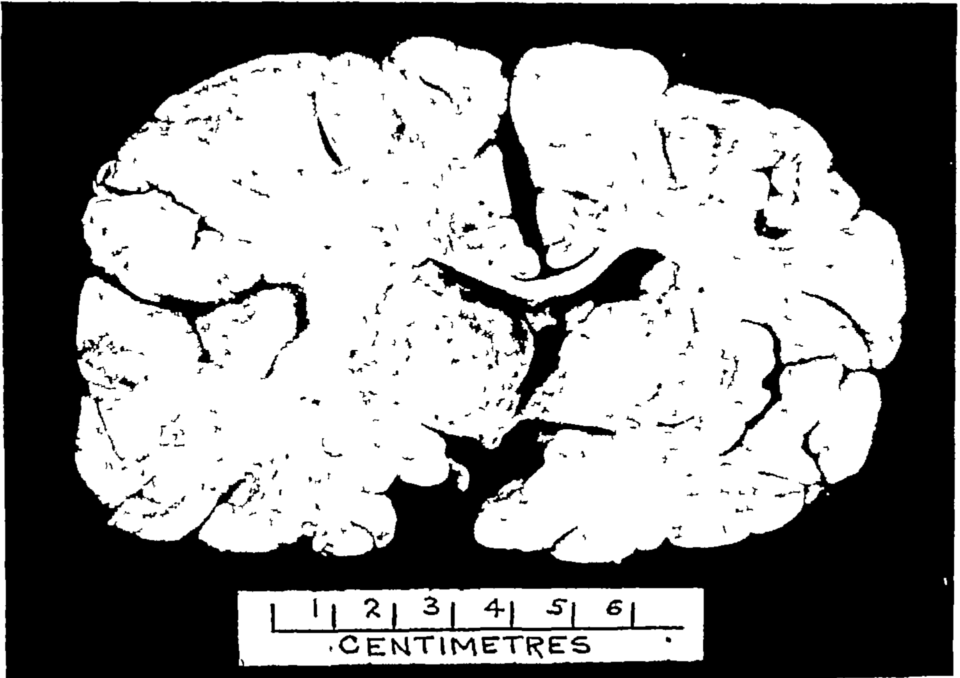


FIG 30
Swelling of the right hemisphere of the brain, due to œdema

and merely aggravates what in any case would have been a fatal injury Alternatively, late meningitis may develop at any stage of the healing process and transform what otherwise would have been a reversible injury into a fatal one. In the early stages, bacteria can gain direct access from the air sinuses to the leptomeninges before natural processes have had time to close the meningeal space by adhesions This explains why it is so dangerous for a patient to raise his intranasal pressure by blowing his nose to remove an obstructing blood clot when a fracture of the anterior fossa is present. Infections are apt to be virulent, not only because the leptomeningeal spaces are widely open but because bruised brain tissue and extravasated blood form a very favourable medium for the proliferation of organisms.

Delayed meningitis in frontal injuries is often secondary to

surface hæmorrhages. In such cases a large vessel has usually been torn. In my experience the vessels concerned in order of frequency are (1) the posterior group of cortical veins draining into the parasagittal sinus, (2) the anterior group of superior parasaggital veins, (3) the meningeal vessels and (4) the dural sinuses. Theoretically all these cases could have been relieved surgically were it possible to diagnose the hæmorrhage and its site of origin early enough.

When a patient survives for twelve hours or more, very probably he has not received a primary injury of the brain which is essentially fatal, and would not succumb if complications did not supervene. Secondary shock, loss of blood, compression or distortion of the brain may render inadequate the circulation in an essential centre, the functions of which are already impaired by a contusion, or they may in themselves be sufficient to cause death. It is with this type of case that the surgeon is particularly concerned, when all the minutiæ of therapeusis, such as the application of the right amount of heat and the giving of the right quantity of fluid, are vital.

After twenty-four hours, pneumonia and meningitis claim a big death roll, and serious injuries in other parts of the body which remain clinically silent are often a contributory cause of death. The importance of injuries to other parts of the body would be more fully appreciated if more detailed post-mortem examinations were made instead of the usual cursory examination of the head. Cases which are recorded as fatal cerebral injuries are occasionally due to high fracture dislocation of the spinal column with cord compression. Fat emboli and other forms of emboli must also occur but are never discovered.

In figures published on large series of cases, fatality rates vary from 8 to 27 per cent.[1] The difference in these figures is difficult to explain. It is partly owing to the differing severity and nature of the injuries, for it is obvious that a simple uncomplicated contusion is less dangerous than a laceration associated with a profuse surface hæmorrhage. In one hospital the Resident Surgical Officer thought it unnecessary to seek expert advice, as he had come to the conclusion that all cases of severe concussion died whatever treatment was carried out, whereas in another hospital a Senior Resident believed that most cases survived if they were left alone

This raises the very important question as to whether conservative treatment, confined to resuscitation from shock, does give results equal to those obtained by more active

[1] Russell Brain, W. "Recent Advances in Neurology," 125 J A Churchill Ltd. London, 1934

cortices produce neural deficits according to their position and extent, but they do not and cannot destroy life directly.

The extent and degree of primary types of injury are determined at the moment of the receipt of the violence and a static form of damage occurs. Such damage may or may not be essentially lethal at the outset. When it is not, secondary developments may supervene and render fatal what otherwise would have been a non-fatal contusion, laceration or diffuse neuronal injury.

Secondary developments are progressive and not static pathological phenomena and, unlike the primary injuries which damage the neurones themselves, they produce their effects by raising the intracranial pressure and embarrassing the cerebral circulation. Also, the chronology of their onset is variable, and each phenomenon by no means occurs in each case. Some degree of shock is usual, and in cases of severe concussion absence of subarachnoid bleeding is rare. Hydrocephalus is usually associated with surface hæmorrhages It not only causes a rise in intracranial pressure but also may act as a local compressive lesion and give rise to localising signs, such as hemiplegia or aphasia. Œdema may be the only complication of a contusion. When it remains local it is probably of no great significance. It may, however, spread rapidly to a whole hemisphere and dislocate the intracranial contents to such an extent that the functions of the brain become disorganised with resulting death. It also causes all kinds of foraminal herniations. Herniations may compress the brain stem at the foramen magnum or at the hiatus tentorii, or they may compress vessels or nerves distant from the site of the primary injury. It will be seen, therefore, that if a patient were co-operative after a severe head injury, the integration of the many pathological states which can affect the brain as the result of acute cerebral trauma is so complex that diagnosis and treatment would still remain a very difficult problem.

Patients moribund from the start who show no sign of mental or physical improvement in spite of treatment usually have received a primary type of injury to the brain which is essentially fatal. Such cases usually die within twelve hours At autopsy, severe lacerations or extensive contusions are often found, and occasionally large areas of the brain are completely pulped. Extensive surface hæmorrhages are often present, but, as far as can be judged, death would have resulted even if these had been absent. In this group of cases nothing useful can be done surgically.

Death may occur within twelve hours from secondary developments alone and particularly from the compression of

CHAPTER III

DIAGNOSIS OF CLOSED INJURIES OF THE HEAD *

DIAGNOSIS[1-7] in acute cerebral trauma of the closed type is usually a difficult problem, since many of the patients resent examination or are profoundly unconscious. Owing to lack of co-operation the customary routine investigation of the nervous system is impossible and much essential information has to be sought by indirect means. Moreover, the neurological picture is often so confused that the whole brain seems to be affected, which, in fact, it is With reasonable care, however, a reliable impression of the nature of the primary brain injury can be obtained and secondary developments diagnosed at a stage when they are still amenable to surgical treatment.

Primary shock must always be treated before a detailed examination is made, not only in observation of surgical first principles but because no trustworthy neurological information can be obtained so long as this state exists A few minutes only are necessary to ascertain the patient's general condition and to determine how deeply unconscious he is and whether there are associated injuries needing urgent treatment. Apart from careful scrutiny of the scalp and body for open wounds, detailed examination may be deferred for half an hour, so that the patient may be warmed up, as he is often admitted to hospital thoroughly chilled.

Examination.—Examinations must be carried out in a warm

* The term "closed" means that the brain has not been penetrated by a missile and that a compound fracture of the vault has not occurred, a fracture of the base opening into the ears or nose may be present

1 Dott, N M "Thompson and Miles Manual of Surgery," 1939, 2
2 Jefferson, G "Discussion of the Diagnosis and Treatment in Acute Head Injuries" *Proc Roy Soc Med*, 1932, **25**, 742
3 Munro, D "Craniocerebral Injuries Their Diagnosis and Treatment" Oxford University Press, 1938, 412
4 Riddoch, G "Discussion of the Diagnosis and Treatment of Acute Head Injuries" *Proc Roy Soc Med*, 1932, **25**, 735
5 Russell, W R "Discussion of the Diagnosis and Treatment of Acute Head Injuries" *Proc Roy Soc Med*, 1932*b*, **25**, 751
6 Symonds, C P "Concussion and Contusion of the Brain and their Sequelæ," "Brock's Injuries of the Skull, Brain and Spinal Cord" Baillière, Tindall & Cox London, 1940
7 Trotter, W "Injuries of the Skull and Brain," "Choyce's System of Surgery," 3, 309 Cassell & Co London, 1932

surgical measures, such as dehydration, spinal drainage or decompression.

To settle this problem I treated a hundred cases conservatively and a comparable series, as far as was possible, actively. The results of this experiment were that fatalities were 30 per cent. higher in the conservatively treated group than in the other. Therefore, in my opinion, not only can many lives be saved by skilled treatment, but sequels can be reduced to a minimum and morbidity rates considerably improved.

Signs referable to the Skull (Figs 31 and 32).—In recent years the importance of damage to the brain has so often been stressed that the older habit of devoting chief attention to the condition of the skull has tended to become overcorrected. Although it is true that what happens to the brain and its covering membranes finally determines whether the patient lives or dies, it must be realised that information obtained from the skull can materially assist in the diagnosis of the nature of the cerebral injury. Moreover, fractures of the paranasal or petromastoid regions, if not diagnosed and treated correctly, lead to meningitis and encephalitis.

When death occurs within twelve hours the brain injury is usually associated with an extensive fracture of the base. In those cases where the signs of injury to the brain are minimal, a fracture of the skull indicates that a considerable force has been applied to the head, and so suitable precautions may be taken, particularly by repeated observation. It is unrecognised cases in this category that are sometimes allowed to go home from casualty departments, only to die from cerebral compression due to an extradural or subdural hæmorrhage. Furthermore, a fracture line crossing the middle meningeal groove will influence a decision in favour of early exploration when an extradural hæmorrhage is suspected but cannot be diagnosed with certainty on clinical signs alone. Even in closed fractures a spicule of bone may pierce the dura mater and lead to traumatic epilepsy if not removed and the dura repaired.

Clinical Signs.—On no occasion must the diagnosis of a fracture of the skull be attempted by the elicitation of crepitus. No useful information is likely to be gained by this method and serious damage may be done thereby. A loose fragment of bone lying outside the dura may be driven inwards to lacerate the brain, or a hæmorrhage may be started which would not otherwise have happened.

The edges of a large depressed fracture can be palpated with certainty, but in small depressions a bony edge can be very closely imitated by the indurated edges of a centrally fluctuating hæmatoma in the scalp, and there are no reliable clinical means of distinguishing between the two. Linear fractures, unless they are widely open, cannot be diagnosed by palpation. Extensive and boggy swellings under the scalp indicative of large subgaleal hæmorrhages always mean that the bone has been broken. Thickening of the temporal muscle and retromastoid bruising are also reliable signs of fracture. On the other hand, bleeding from the nose or ears often comes from laceration of soft tissues Profuse and persistent bleeding from these sources is suggestive of fracture, but the only incontrovertible evidence that the skull has been

room and under good lighting. When these two conditions have been obtained, all the covering bedclothes should be removed and the patient completely stripped, otherwise it is often impossible to get a true perspective of the complex problem presented and important signs may be overlooked. First of all, spend a few minutes purely in inspection · analyse the posture of the patient and observe his spontaneous movements . watch his facial expression and note his colour and type of breathing. When this has been done, make sure that there are no fractures of the limbs which require temporary splinting to prevent them becoming complicated or compound if the patient is, or should become, restless. From the previous chapter it will have been learnt that serious injuries to the chest, spine and abdomen are by no means uncommon Therefore the abdomen should be palpated for rigidity and percussed for the presence of free fluid or air in the peritoneal cavity, the latter being indicated by loss of liver dullness. The chest wall should be palpated for fractured ribs and the chest cavity percussed and auscultated for pneumothorax or hæmothorax. Also, a finger should be run along the spine to detect the possible irregularity of a fracture dislocation.

If these things are not done a reliable estimate of prognosis is impossible and treatment of an injury elsewhere in the body may be omitted, which is just as important as that directed to the damaged cerebrum.

When associated injuries have been noted and their severity assessed, the head should then be examined for signs referable to injury of the skull, and these should be kept separate from those referable to the brain, otherwise a jumble of confusing information will accrue

The hair is often matted with blood, and this must be washed cleanly away if the scalp is to be examined adequately and possible compound fractures eliminated. In any case, wounds of the scalp not accompanied by underlying fractures are important, because if not correctly treated they may lead to fatal or troublesome septic complications The face should be cleaned and abrasions treated so as to minimise subsequent infection. Pools of blood must be dabbed from the ears, otherwise it is impossible to know whether the bleeding comes from a fractured base or has merely trickled into the external auditory meatus from a wound in the soft tissues. Finally, attention should be directed to those signs which are referable to the cerebral injury, and this is by far the most difficult part of the examination and one which necessitates a great deal of practical experience as well as a knowledge of theoretical neurology. Restlessness is never a sufficient reason for omitting to make a detailed examination.

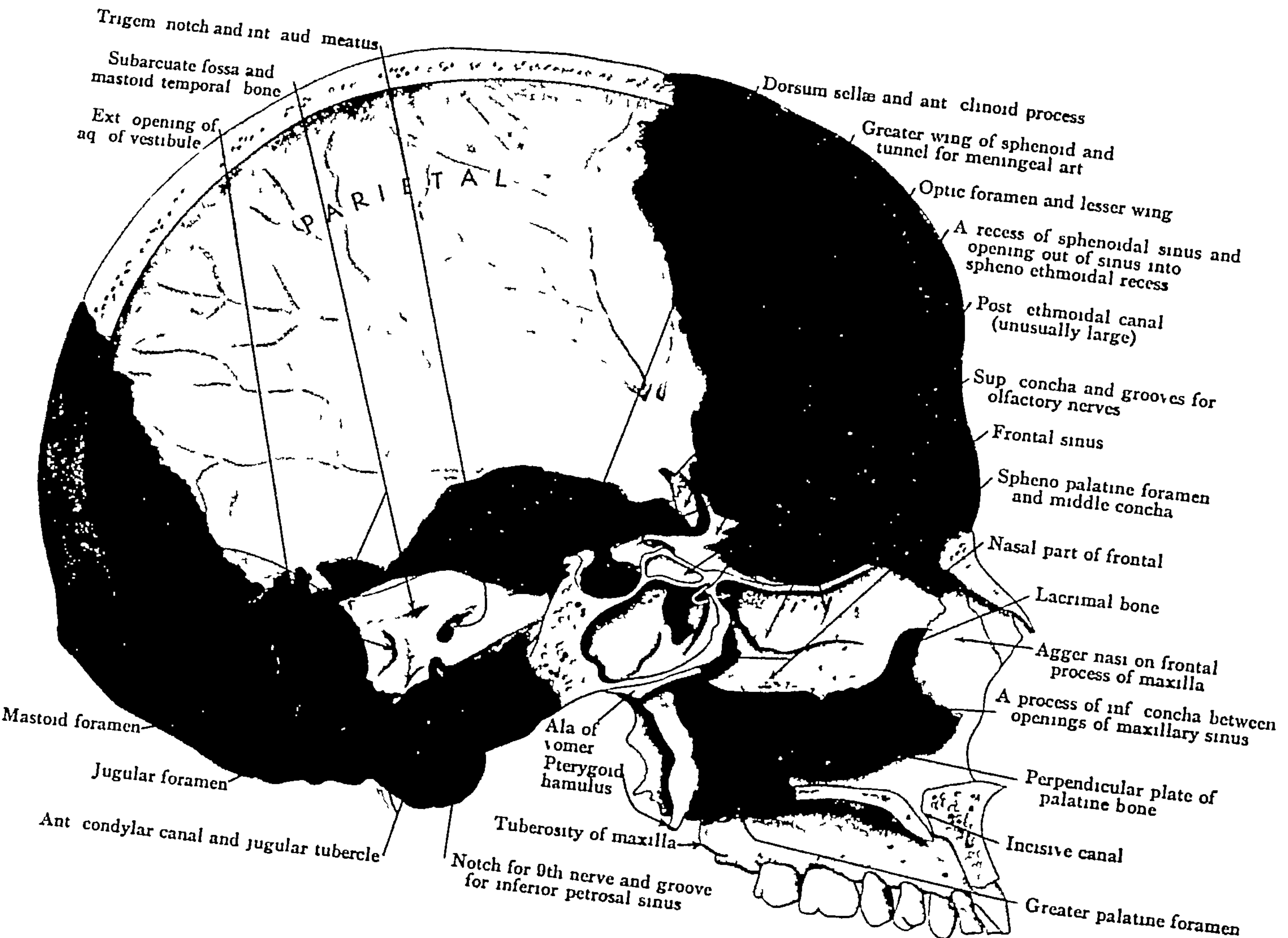


Fig 32

Sagittal section of skull (E B Jameson)

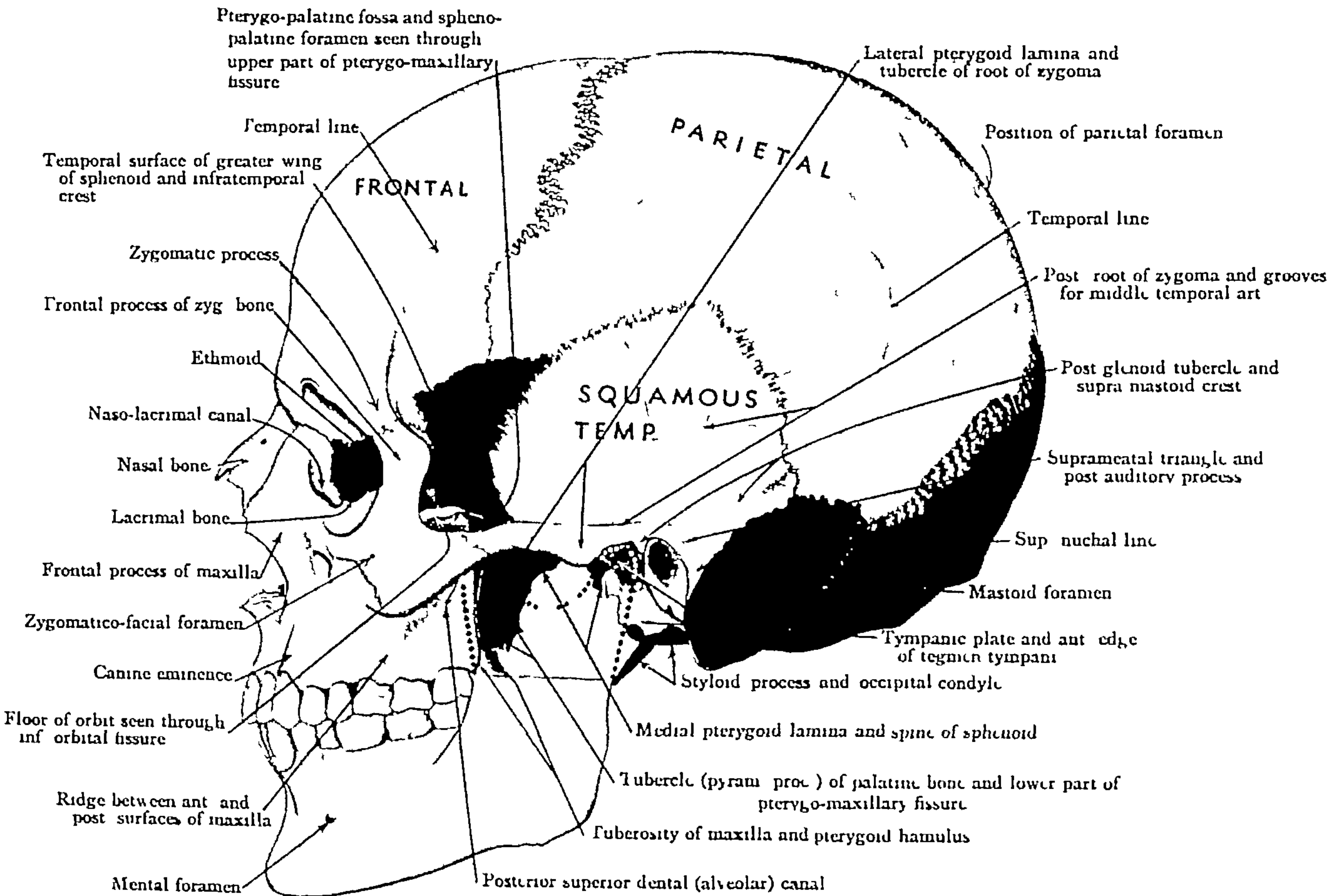


Fig 31

Side view of skull (*E B Jameson*)

In closed head injuries three routine views are necessary: (1) anteroposterior, (2) right lateral and (3) left lateral. A tangential view should be added when a depressed fracture is present or suspected.

The plane which runs through the centre of each external auditory meatus and which cuts the lower margins of the orbit is the radiographic base from which the various accepted projections are made. It is known as the orbitomeatal plane (Fig. 34).

1. *Anteroposterior View.*—The patient is placed on his back and the head manipulated so that the orbitomeatal and sagittal

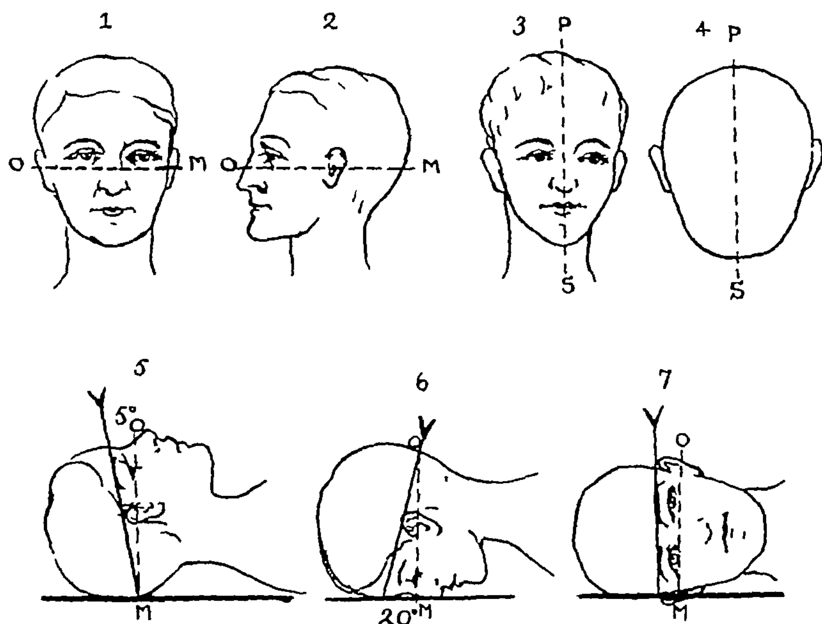


FIG 34

Radiological positions

1 and 2, The orbitomeatal plane 3 and 4, The parasagittal plane 5, The anteroposterior view 6, The postero-anterior view 7, The right lateral view "V" indicates the axis of projection of the X-rays

planes of the skull are exactly perpendicular to the floor. If the sagittal plane is allowed to rotate to one side an asymmetrical view of the skull will be obtained which is difficult to interpret. The rays are then projected so that they make an angle of 5° with the orbitomeatal plane (see Fig 34). The object of angulating the X-ray tube is to bring the orbits low on to the films so that they do not obscure large areas of the vertex. The frontal bone can be seen in greater detail by a postero-anterior than by an anteroposterior view, but this necessitates turning the patient on to his face, a position he will rarely tolerate.

2. *Lateral View.*—In lateral views it has been agreed that the side of the skull nearest to the X-ray film shall determine which is the right and which is the left lateral shoot, *e g.*, the right side down is the right lateral view. Great care must be taken to obtain

broken is the presence of cerebrospinal fluid or brain tissue in the discharge

Subconjunctival hæmorrhages are significant of fracture only (1) when they cause œdema of the conjunctiva, (2) when they are so extensive that it is impossible to see beyond their posterior limits in any position of the eye (Fig. 33) or (3) when they are so large that they displace the eyeball and restrict its movements. Finer details than these of differentiation between the hæmorrhages of a fracture and a "black eye" are of little practical value since



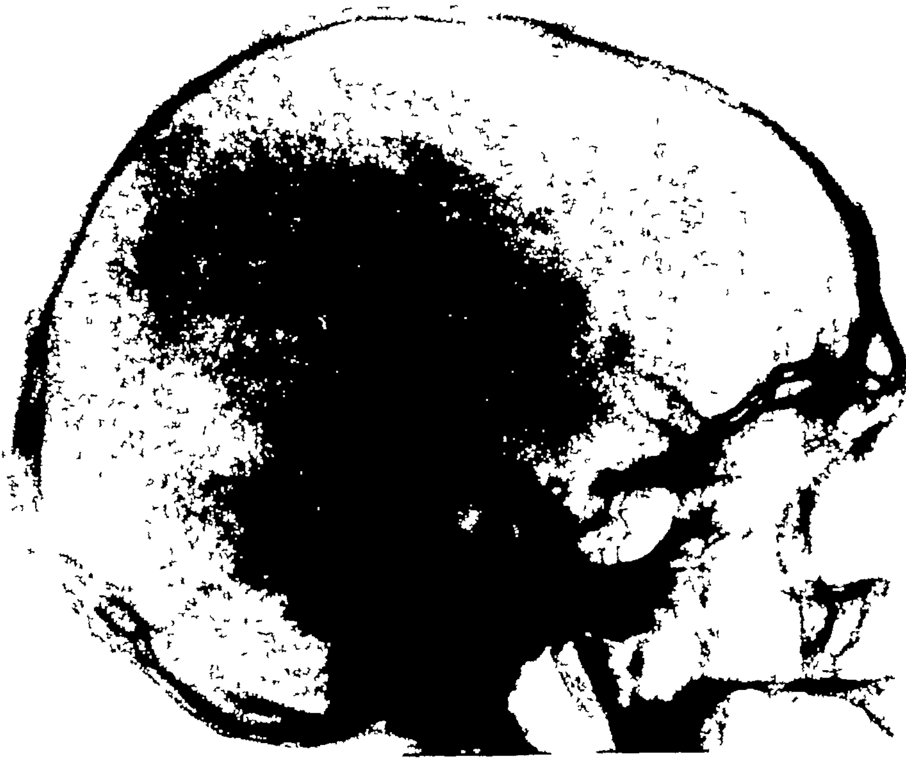
(a) (b)

Fig 33

a, This type of intra-orbital hæmorrhage is indicative of fracture of the anterior fossa
b, A flame-shaped hæmorrhage is caused by contusion of the soft tissues and not by fracture

a fracture of the base may not lead to an intra-orbital hæmorrhage, but may be and usually is associated with a black eye Gross deformities of the skull are incompatible with life.

Radiography.—Since the nature of a cranial injury can aid in the diagnosis of the underlying cerebral state, radiography should be used as a routine measure in the acute stages of cerebral trauma. Usually it is best to take the photographs immediately at the end of the clinical examination, as clinical findings will determine the views that may be necessary Unfortunately the decision as to whether radiography should be used is often determined by convenience rather than by conviction, or by someone other than the surgeon, and these faults ought to be corrected. It is unwise further to increase shock by transporting the patient to the main X-ray department of the hospital for detailed examination, but there is no reason why he should not be examined in his bed by means of a portable apparatus. With reasonable care, satisfactory films can be obtained Useless films are usually due to faulty exposure rather than to misbehaviour of the patient With sufficient help and restraint the head of the patient can be manipulated into the desired position and no damage need occur to the apparatus



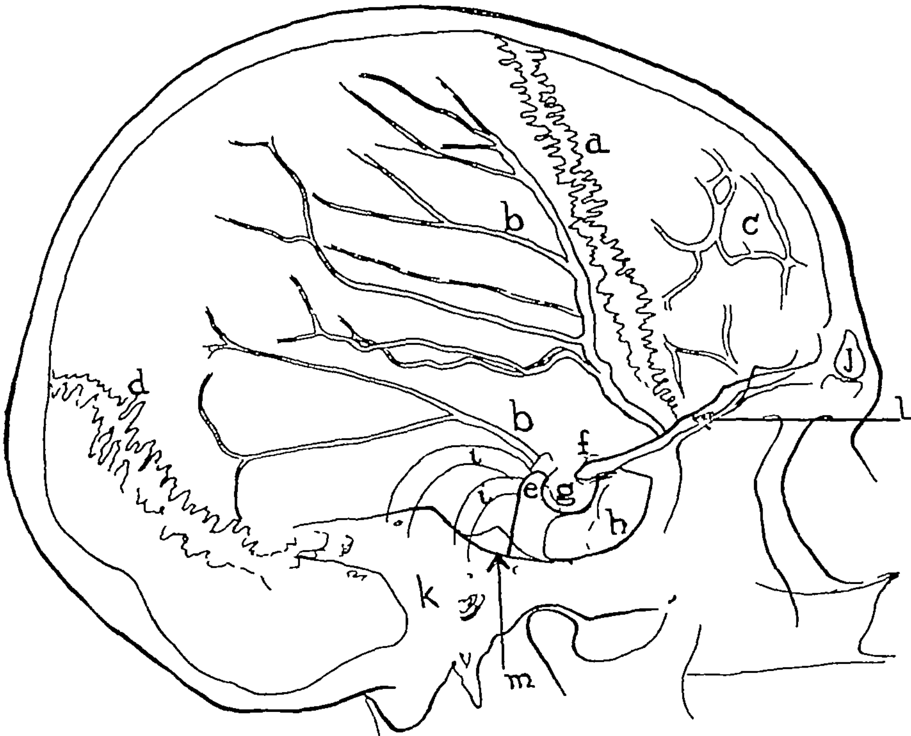


Fig 35

The normal markings on the skull (lateral view)

a, Frontoparietal suture, *b*, meningeal grooves, *c*, diploic channels, *d*, occipitoparietal sutures *e*, posterior clinoid process, *f*, anterior clinoid process, *g*, pituitary fossa, *h*, sphenoidal air sinus, *i*, shadows of the ears, *j*, frontal air sinus, *k*, petromastoid bone, *l*, floor of anterior fossa, *m*, floor of middle fossa

a true lateral view, otherwise the known landmarks of the skull become obscure. The sagittal plane must be parallel with, and the orbitomeatal plane perpendicular to, the ground (see Fig. 34). These conditions are most easily obtained with the patient on his face, but owing to restlessness the picture usually has to be taken with the patient supine, in which case the head must be forcibly turned to one side and the opposite shoulder raised on a pillow. When the patient vigorously refuses to have his head put into this position, the rays may be projected parallel to the floor with the patient's nose pointing upwards and with the film casette held against the opposite side of the head.

3. *Tangential View.*—In tangential views the head is so oriented that the suspected depression lies end-on to the projection of the X-rays.

Special views are necessary to demonstrate certain types of injury about the mastoid bones, petrous bones, paranasal air sinuses, and optic foramina, but as these examinations are rarely urgent, they may be deferred until the patient is well enough to be taken to the main X-ray department of the hospital. The radiological technique required for these various examinations has been described by Cairns and Jupe [1]

As a linear fracture may easily be confused with the natural markings in the skull, a differential diagnosis of the various radiological features will be found in the following table (Figs 35-40) [2] A simple fracture line may remain visible for six months in children and for three years in adults.[3, 4]

DIFFERENTIAL DIAGNOSIS OF VARIOUS RADIOLOGICAL FEATURES OF SKULL

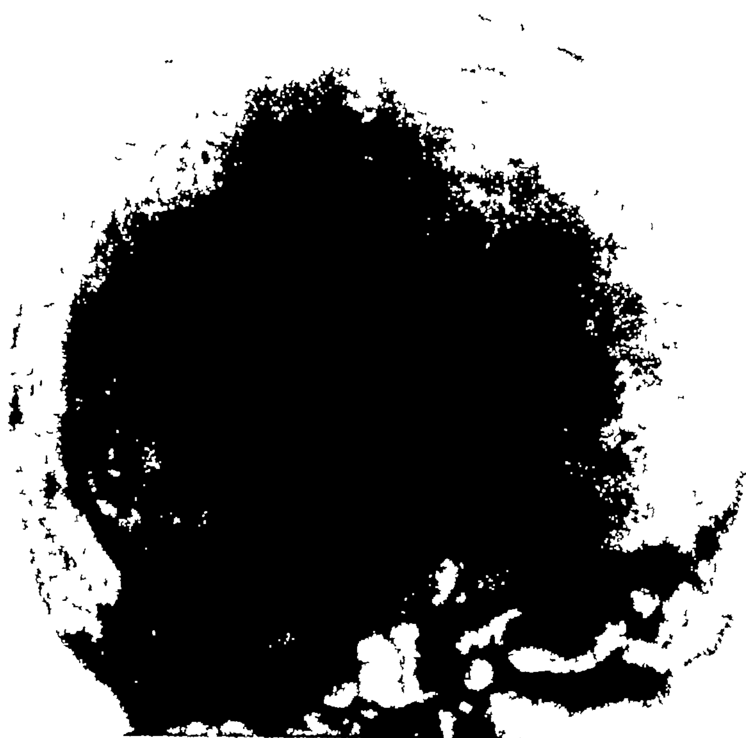
Fracture Lines	Suture Lines	Meningeal Grooves	Diploic Channels
Clean - cut edges Run in all directions May cross suture and arterial lines Change direction abruptly Branch irregularly	Fine or dentate lines Run in constant positions May be widened by trauma or hydrocephalus	Fairly sharp margins Run in known directions Branch dichotomously Calibre diminished from below upwards	Fairly sharp margins Change course abruptly and form irregular patterns Often start in lakes near the sup long sinus Vary in width and often are beaded in appearance

[1] Shanks, S C, Kersley, P, and Twining, E W "A Textbook of X-ray Diagnosis" Lewis & Co London, 1938

[2] Wakeley, C P G, and Orley, A "A Textbook of Neuro-radiology" Baillière, Tindall & Cox London, 1938

[3] Stewart, W H "The Time Factor in the Disappearance of Roentgenographic Evidence of Fractures of the Skull" *Brit Jour Rad*, 1925, **30**, 399

[4] Vance, R G "The Healing of Linear Fractures of the Skull" *Amer Jour Roentgen*, 1936, **36**, 744



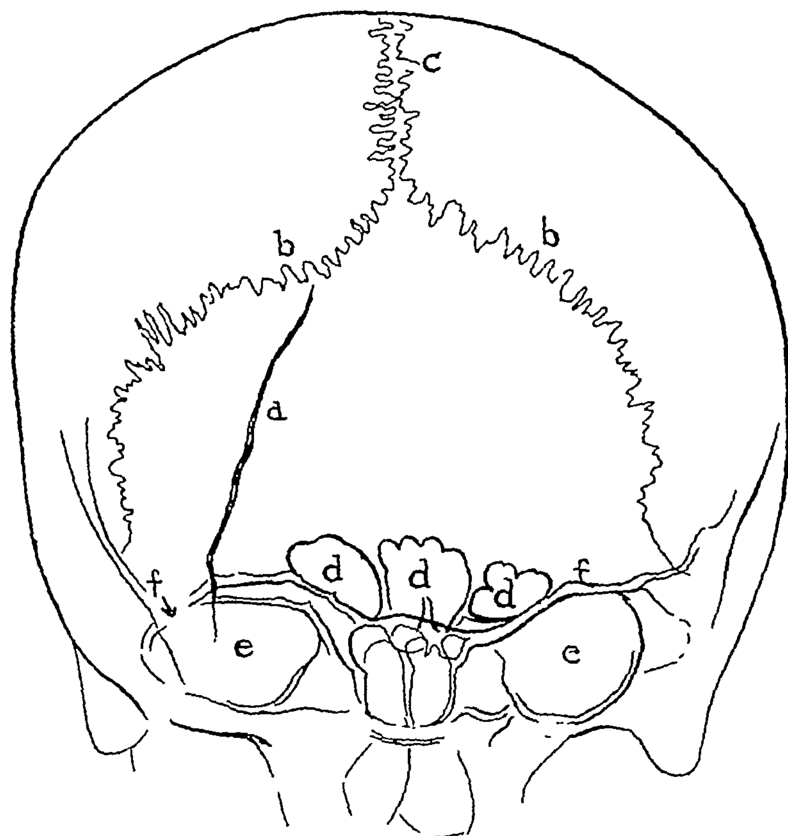


Fig 38

The normal markings on the skull (anteroposterior view)

a, Fracture line, *b*, occipitoparietal sutures, *c*, intraparietal sutures, *d*, frontal air sinus *e*, orbits, *f*, floor of anterior fossa



FIG 36—Diploic channels may closely resemble fracture lines and are often mistaken for them This skull has not been fractured



FIG 37—Stellate fracture lines Compare these with the meningeal grooves and diploic channels, both of which can be plainly seen

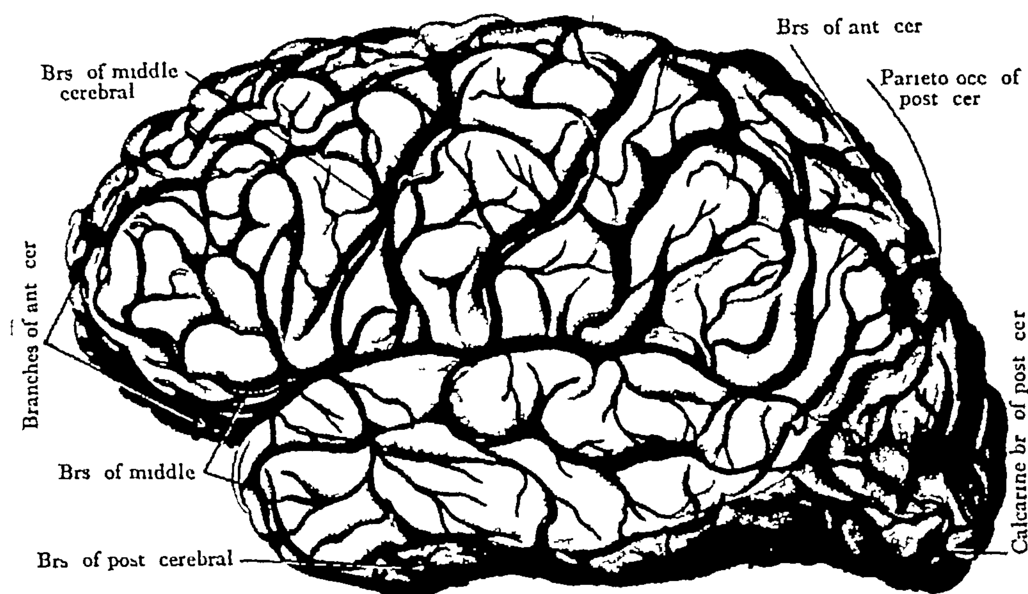


Fig 41
Arteries of superolateral surface (*E B Jamieson*)

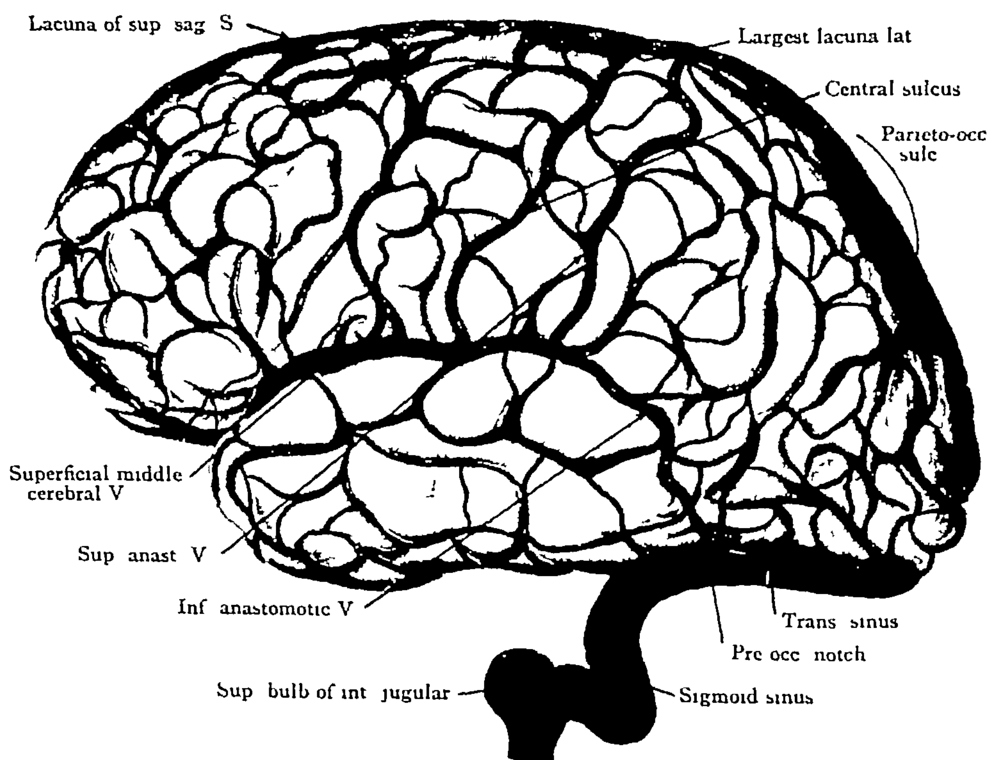


Fig 42
Veins of superolateral surface, and venous sinuses (*E B Jamieson*)

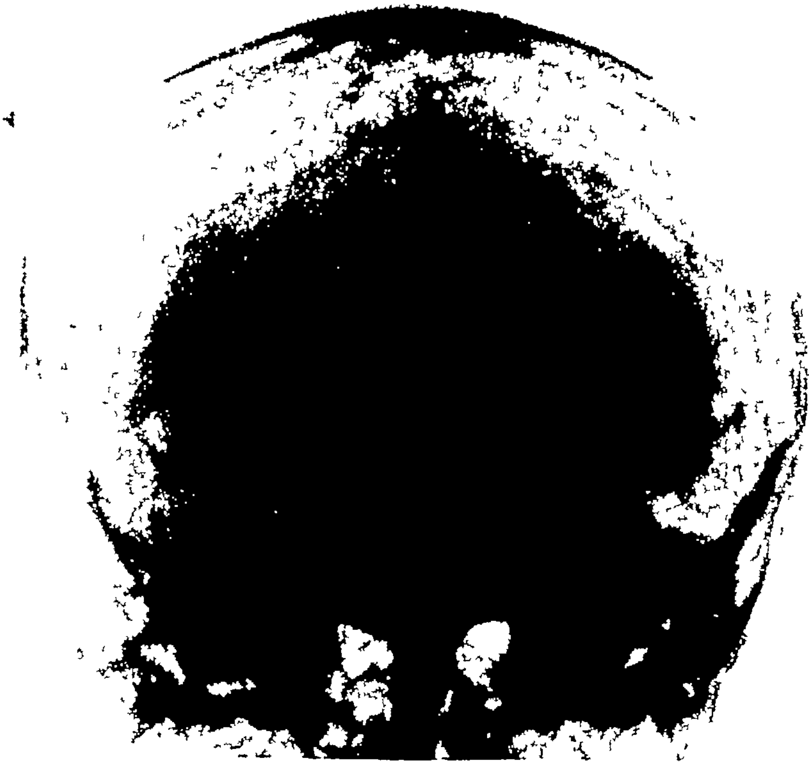


Fig 39 —"Springing" of the occipitoparietal suture This is equivalent to fracture



Fig 40 —The fine linear type of fracture which is so commonly over looked In a poor film it would have been overlooked

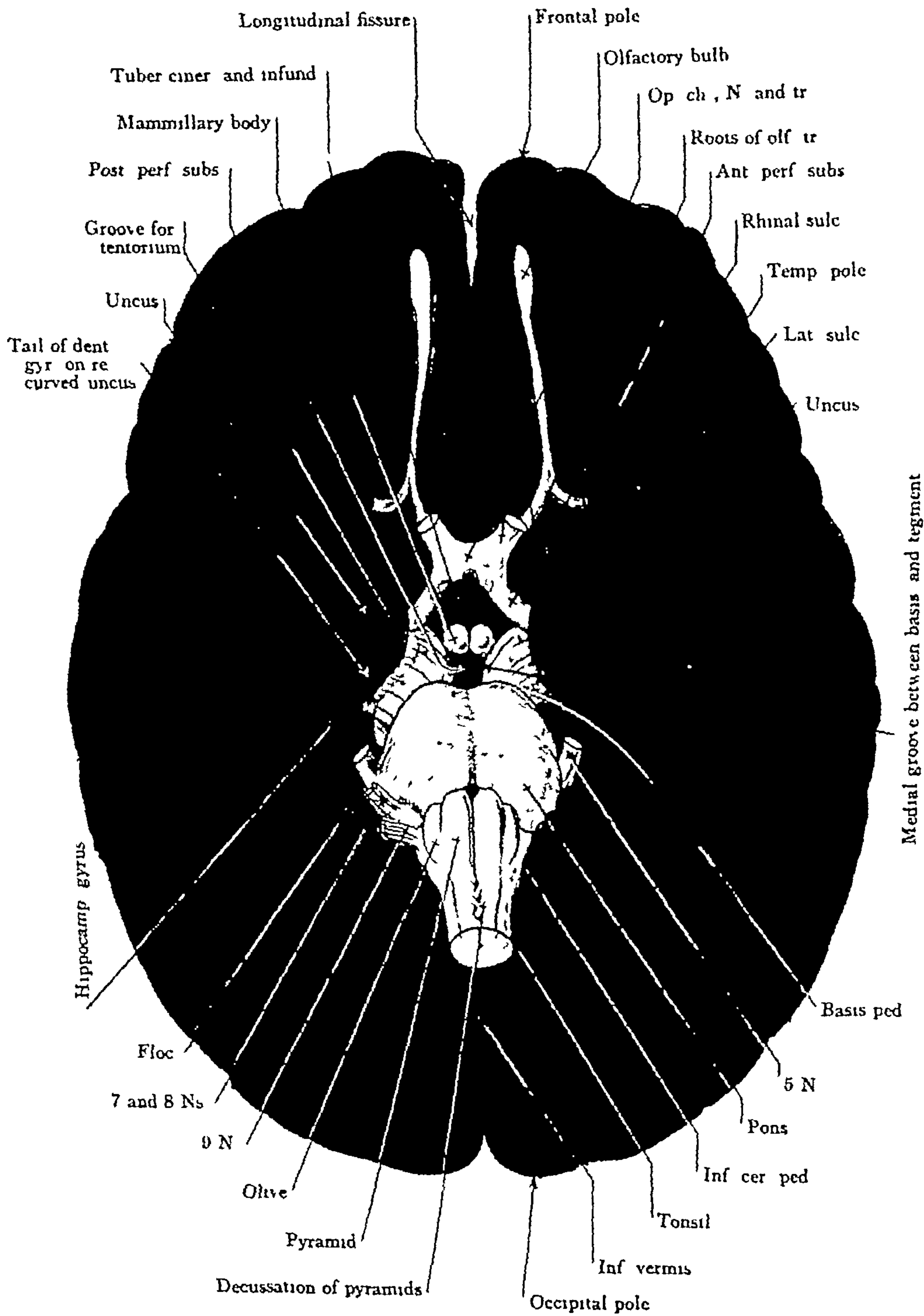


FIG 44

Base of brain (Brain hardened *ex situ* in order that the basis pedunculi might be seen) (*E B Jamieson*)

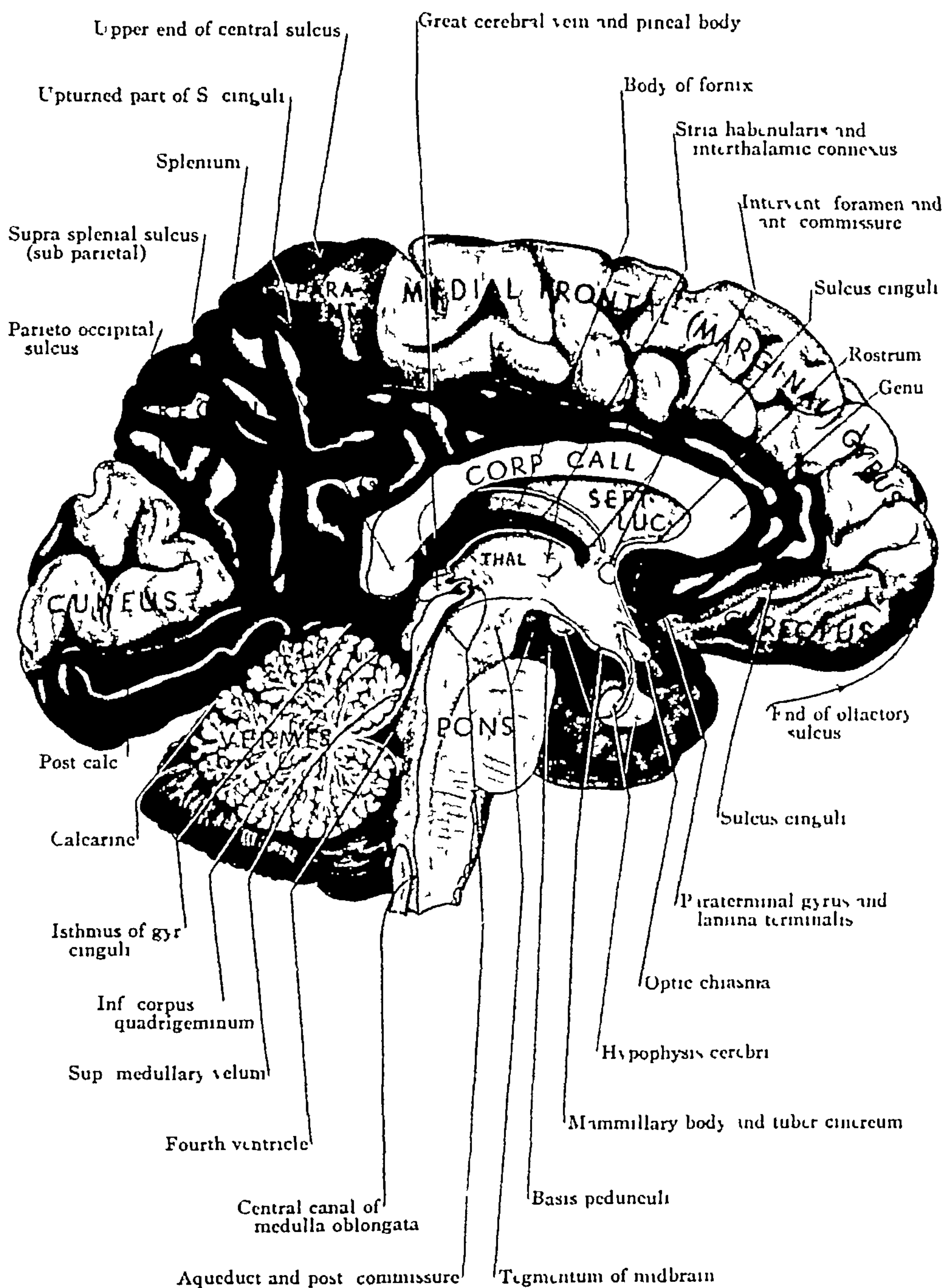


FIG 43

Median section of brain (*E B Jameson*)

problem than in non-traumatic neurological cases, because the dominant features of the illness—unconsciousness and restlessness—have no precise localising value and no definite pathological significance. Moreover, there are no clinical syndromes which can with any certainty be attributed to contusion and laceration. The onset of the cerebral state is so rapid that it cannot be analysed

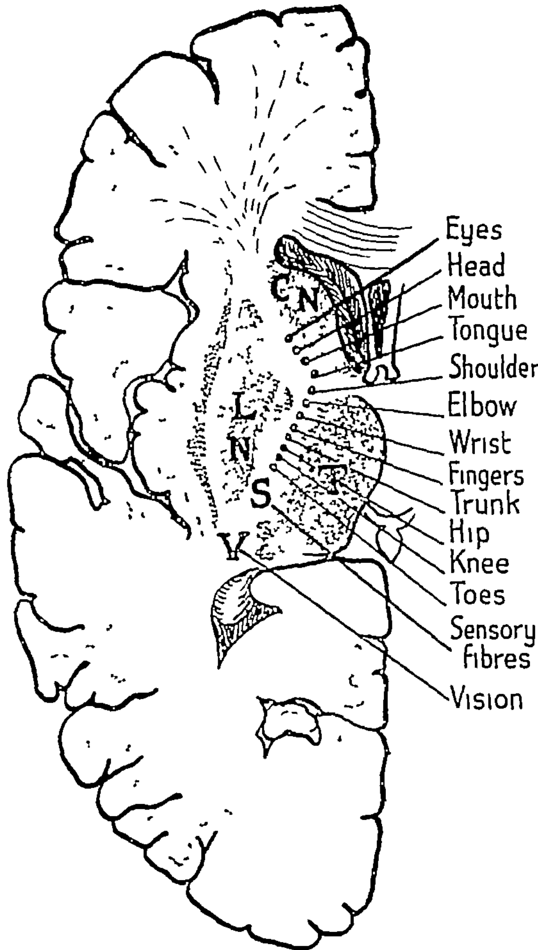


FIG 46

The disposition of the cortical projection fibres at the level of the internal capsule

CN, Caudate nucleus, LN, Lenticular nucleus, T, Optic thalamus

save on those rare occasions when a latent interval occurs, and the illness may take an extremely variable course according to the development of secondary phenomena Also, ventriculography and encephalography are, by the nature of the lesion, not so useful in traumatic as in non-traumatic states In many of the more serious cases their dangers preclude their use.

It would therefore be wrong to attempt to oversimplify diagnosis in acute cerebral trauma as so many people have done in the past, for if it is not realised that the problem is essentially

Signs referable to the Brain (Figs. 41-44).—Although the physiological functions of each part of the brain are so closely integrated that derangement in any one part makes its effect felt in all the others, certain nervous activities are dominantly represented in relatively circumscribed areas of the brain tissue (Figs. 45 and 46). If this were not true, anatomical localisation in diseased states would not be possible

In non-traumatic neurological cases the pathological change is usually discreet enough to permit of precise anatomical localisation

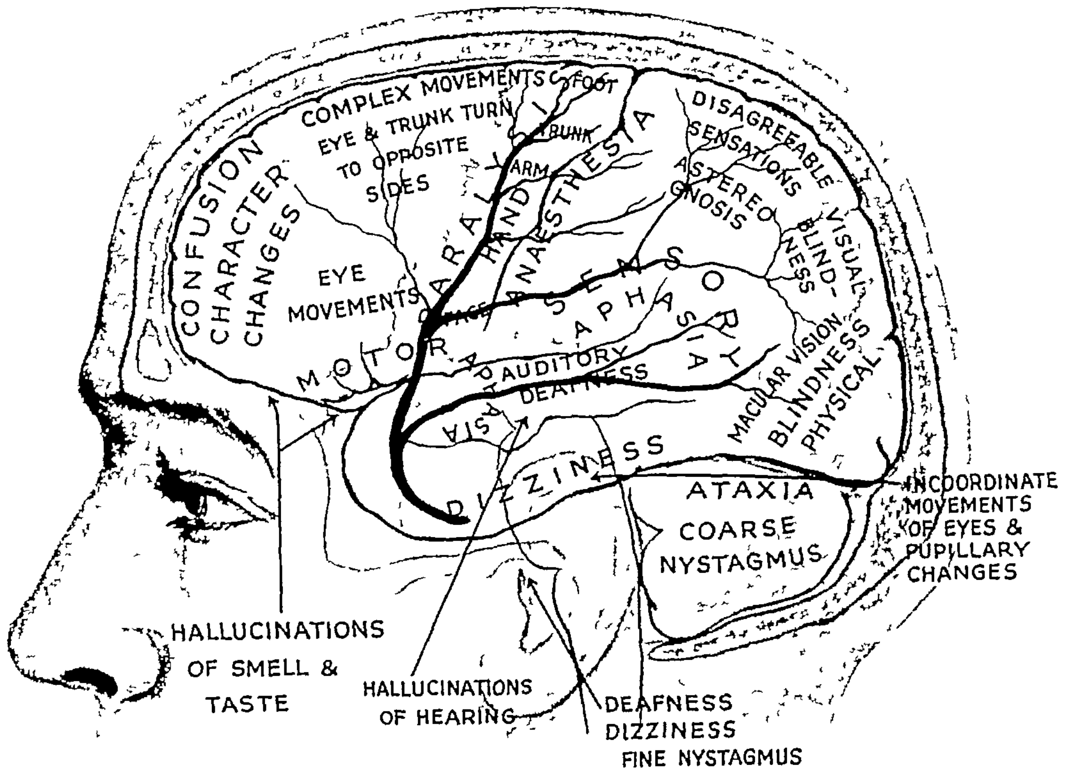


Fig 45
The localisation of function in the cortex of the brain

on clinical grounds alone Diagnosis of the nature of the lesion, on the other hand, is rather more difficult, and is deduced from the mode of onset of the illness, from its progress and from the combination of symptoms and signs which are present at any particular phase in the life-history of the disease. Also, pathological diagnoses may be aided or confirmed by cytological, chemical and bacteriological examination of the cerebrospinal fluid, and by special investigations such as spinal manometry, encephalography or ventriculography

In traumatic cerebral neurology, anatomical localisation and the diagnosis of the nature of the lesion is a much more difficult

degrees of confusion, and these may be graded according to the responses that can be obtained to commands. In order to obtain some kind of uniformity in the assessment of confusion the Medical Research Council[1] have suggested that the following classification be adopted —

Mild.—A state in which the patient, though presenting the characteristic feature of confusion in some degree, is capable of coherent conversation and appropriate behaviour.

Moderate.—A state in which the patient, though out of touch with his surroundings, can be got to give relevant answers to simple questions, such as "What work do you do?" "How old are you?" "Where do you live?"

Severe.—A state in which the patient, though for the most part inaccessible, will occasionally show adequate response to simple commands forcibly given and, if necessary, reinforced by appropriate gestures, *e.g.*, "Put out your tongue," "Take my hand."

The poles or association areas of the frontal lobes are thought to be concerned chiefly with the higher grades of the intellect and with the control of emotion, but they are not, as is so often believed, responsible for all kinds of thinking and neither are they the parts of the brain in which consciousness is centred

So far, electrical or any other kind of physical or chemical stimulation of the cortex of the frontal poles has not yielded any useful information as regards the intellect, and what knowledge we have of the functions of the frontal association areas has been obtained from the effects either of pathological lesions in these areas or of lobectomies made in the operative removal of tumours. Unilateral resections of that part of the brain which lies within the anterior fossa of the skull either on the left or right side do not lead to obvious intellectual changes as Penfield[2] and Jefferson[3] have shown, apart from slight deterioration of initiative Bilateral lobectomies, on the other hand, lead to a subnormal mentality, though according to Brickner,[4] who made exhaustive tests on one patient in whom both frontal poles had been removed, there are no specific functions in the frontal association areas which are not present to some degree elsewhere in the brain

In view of the above findings it is reasonable to suggest that

[1] Medical Research Council "Glossary of Psychological Terms commonly used in Cases of Head Injury" M R C War Memorandum No 4 March, 1941

[2] Penfield, W G, and Evans, J "The Frontal Lobe in Man a Clinical Study of Maximum Removals" *Brain*, 1935, **58**, 115

[3] Jefferson, G 'Removal of Right or Left Frontal Lobes in Man" *Brit Med Jour*, 1937, **2**, 199

[4] Brickner, R M "The Intellectual Functions of the Frontal Lobes," **16**, 354 The Macmillan Co New York, 1936

complex, bewilderment and not information will be the final result of most clinical examinations.

Confusion and Unconsciousness.—The most important feature of any closed injury to the brain is unconsciousness. It is a state of extreme gravity, and as long as it exists the patient's life is in danger. When it comes on immediately after accident it is due either to diffuse neuronal injury or to damage to the ganglia of the brain stem, of the thalamus or of the hypothalamus, although it may be perpetuated by a secondary compression as the effects of the primary injury recede. A period of consciousness immediately following the accident, however short, as a rule means that any later ensuing state of unconsciousness is due to secondary developments, such as an increasing hæmorrhage, which can be cured by surgical means The slightest change in the depth of unconsciousness either one way or another is an infallible sign of improvement or retrogression, and as recovery of consciousness is a gradual process, whether this be rapid or slow, repeated examinations at frequent intervals are necessary if changes are to be recognised early.

Depth of unconsciousness may be judged by the reactions of the patient to external stimuli and may be classified thus —

Coma —This is a state of complete unconsciousness in which there are no psychologically understandable responses either to external stimuli or to inner needs. Certain primitive responses, such as the corneal, swallowing or tendon reflexes may or may not be present, and their absence is indicative of a very serious state within the lower centres of the brain The patient cannot be roused or be compelled to make a movement by any kind of verbal command or by the infliction of pain, such as pressure on the testicle or of pricking of the finger-tips.

Semicoma —In this state there is a complete lack of co-operation. The patient, however, can be made to make some kind of movement or to change his expression in response to painful or disagreeable stimuli A test I have found most useful is to push the angle of the jaw forwards forcibly and to hold it in this position for a few moments Lack of response may be taken to mean coma When this manœuvre causes obvious discomfort, the patient is no more than semicomatose All the primitive reflexes, such as swallowing and closure of the eye on stimulation of the cornea, are present In coma there is retention of urine, possibly with overflow, whereas in semicoma the bladder empties reflexly whenever it becomes distended

Confusion —By confusion is meant a clouding of consciousness In this state the patient obviously makes an effort to think, but is unable to do so with any clarity or speed There are, of course,

degrees of confusion, and these may be graded according to the responses that can be obtained to commands. In order to obtain some kind of uniformity in the assessment of confusion the Medical Research Council[1] have suggested that the following classification be adopted :—

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[2] Penfield, W. G., and Evans, J. " The Frontal Lobe in Man: a Clinical Study of Maximum Removals." *Brain*, 1935, **58**, 115.

[3] Jefferson, G. " Removal of Right or Left Frontal Lobes in Man." *Brit. Med. Jour.*, 1937, **2**, 199.

[4] Brickner, R. M. " The Intellectual Functions of the Frontal Lobes," **16**, 354. The Macmillan Co. New York, 1936.

confusion following cerebral trauma may occasionally be due to contusion of the frontal lobes and is a localising sign of injury to these regions, whereas states of deeper unconsciousness, such as semicoma and coma, are indicative of a diffuse non-localisable neuronal injury.

Posture and Movements.—There is little doubt that the functions of all the parts of the motor cortex are very closely associated with each other, and this leads us to a consideration of the integration of the nervous system as propounded by Hughlings Jackson, who is regarded as the father of modern neurology. Basing his opinions largely on the activities of the motor system, Jackson came to the conclusion that the nervous system was built up in three different levels. The lowest concerns movements of the simplest form which are governed by the centres in the spinal cord and lower parts of the medulla In Walshe's[1] words:

"The excitable motor cortex of the physiologist is the seat of the 'middle level' of the motor function. It has been evolved out of the lowest level and in it are represented, or reinco-ordinated, the simple and general movements represented in the lowest level. The re-representation consists in the synthesis of complex and special movements of the lowest level, and the experimental observations of Leyton and Sherrington have lent detail and confirmation to this conception. For Jackson, the 'præfrontal region' is the seat of his highest level Here there takes place a further analysis and synthesis of movements represented in the middle level into the most complex and special movements of which the organism is capable.

"There are no 'abrupt' localisations in these two levels and *all* movements are widely represented throughout them"

If I may be allowed simpler terms, the lowest centres are where the pattern of each section of the movement machine is moulded and the excitable motor cortex where the whole machine is assembled. The præfrontal region represents the man who works the machine and makes it do the work for which it is intended.

As a higher centre is eliminated by injury, a lower functional level is released and becomes the integrating centre of the activities in the neurones below (Fig 47). When the basal ganglia are eliminated there is poverty and slowness of movement, rhythmic tremor, rigidity, a fixed facial expression and diminished tendon reflexes Release of the system governed by the red nucleus leads to contraction of those muscles which are concerned in maintaining the upright posture and to relaxation of the antagonists of these groups. Postures determined by red nuclear influences are affected by proprioceptive impulses from the muscles of the neck and

[1] Walshe, F M R "Syndrome of Premotor Cortex" *Brain*, 1935, **58**, 75

from the labyrinth. These are known as the righting reflexes, and are so designed that the limbs and body are moved into the position of optimum balance according to movements and positions of the head. Any lesion which cuts off the descending tracts from the red nucleus but leaves Deiter's nucleus intact leads to decerebrate rigidity. In this state all the muscles of the body and

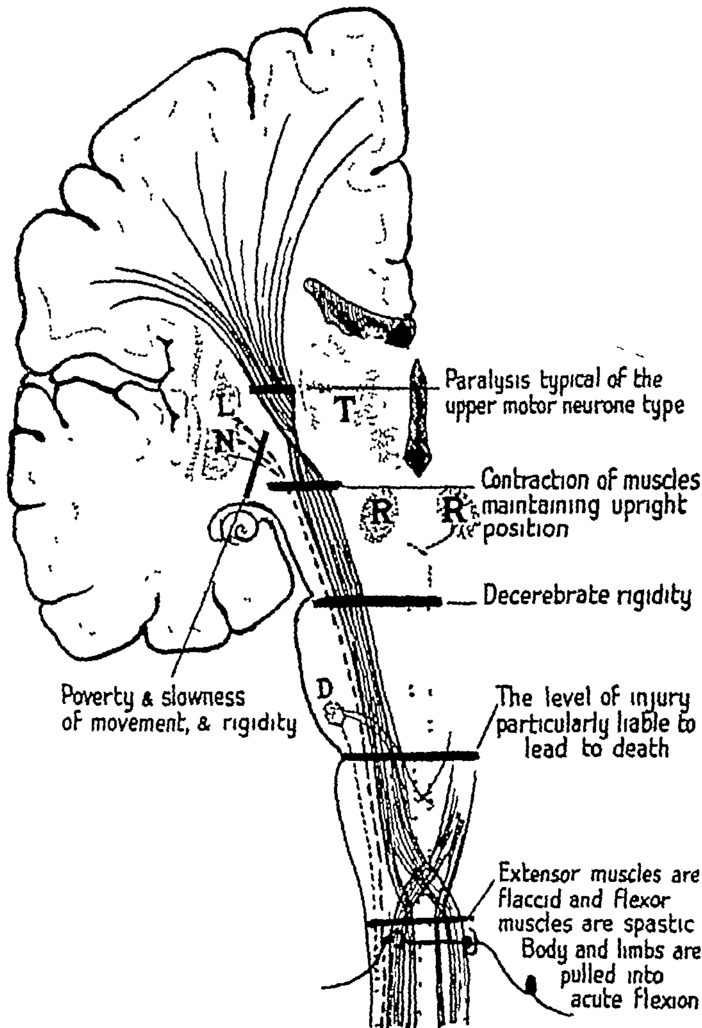


FIG 47

The pyramidal and extrapyramidal pathways According to the level of interruption in these pathways different nervous phenomena ensue This is one of the main reasons why the clinical pictures in head injuries are so variable

LN, Lenticular nucleus, T, Optic thalamus, R, Red nucleus, D, Deiter's nucleus

limbs become rigid, so that the legs and arms act as props. In man the hips and knees are fully extended and the ankles plantar flexed. Sustained muscle spasm resists any attempt at passive movement and reflexes cannot be elicited. The arms may be extended or flexed and they are held firmly in this position by agonist and antagonist muscle groups, occasionally the body may be drawn into a position of opisthotonos. Changes in position of

the limbs, and particularly of the arms, may be brought about by changing the position of the head, the requisite impulses in this case coming from the labyrinths.

When the centres below Deiter's nucleus take control, the extensor muscles become flaccid and the flexor muscles go into spasm, with the result that the limbs and body are pulled into acute flexion

The above neurological phenomena have been mentioned because they help to unravel the meaning of those peculiar postures and movements which are so often seen in the early phases of cerebral trauma and which cannot be explained by a superficial injury to the precentral area. So often clinical observers unsuccessfully attempt to explain paralyses and postures purely by reference to the motor cortex where they know that the body is represented upside down with the hand to the mouth and with the leg over on the mesial side of the brain.

After a severe head injury a patient may lie on his back with his jaw dropped and with his flaccid limbs taking up positions determined by gravity. Alternatively he may be curled up on his side and resent interference Between these two postures any kind of position and type of movement may be seen. Completely flaccid limbs and a fallen jaw usually mean that the whole nervous system is in a state of severe shock, and if improvement does not rapidly take place the patient will die A patient who has been only slightly dazed will look as though he is asleep in the ordinary way His position will be comfortable and the tone in his facial muscles will be good. Prognosis is usually favourable when a patient lies curled up on his side and resents being moved from this position.

Decerebrate rigidity is a most important sign. When it occurs immediately after an injury it means that the upper part of the brain stem has been contused, but when it develops after an interval it is due to a tentorial herniation, the result of some secondary development such as œdema or a massive hæmorrhage This state may suggest the onset of meningitis, particularly if the head is drawn backwards or if the temperature rises, and both these signs may be present in decerebrate rigidity. A lumbar puncture should never be done to establish a diagnosis, as removal of cerebrospinal fluid from the spinal theca will lead to further impaction of a pressure cone. Differential diagnosis is usually not difficult, since in decerebration the patient lies still and the muscle rigidity is persistent, whereas in the early stages of meningitis the patient is often restless and muscle tone variable

Except in coma, movements of all four limbs are usually spontaneous or can be produced by suitable stimuli. Muscle

tone is often changeable, so that at one moment a joint can be manipulated easily through a full range of movement, whereas at the next the muscles may be in a state of spasm. Reflexes are also variable The knee jerks may or may not be active, and the plantar response may fluctuate from flexor to extensor at short intervals. These observations point to damage of the motor pathways, but unfortunately they are of no precise localising value and are best explained by rapid changes of the circulation within the areas concerned.

A paralysed limb lies motionless in spite of suitable stimuli. In the early stages of paralysis due to any cause, the muscles are flaccid and the limb falls heavily to the bed with a slap when raised and allowed to drop without support. Comparison with the fall of a better-controlled non-paralysed limb will demonstrate this flaccidity even more closely

Convulsive seizures or epileptic twitchings in any group of muscles localise the injury to the opposite Rolandic cortex They may indicate a contusion or irritation by a subdural or extradural clot, although on clinical grounds it is impossible to differentiate between the two.

Hemiplegias are much more common than monoplegias, and they may occur immediately or develop after an interval. A hemiplegia immediately following an injury is best regarded as due to bruising of the motor cortex, especially in children. Even if the diagnosis proves to be incorrect, no valuable time will have been wasted as regards recovery of power if an operation has to be performed later. Interval hemiplegias may be caused by (1) subdural or extradural hæmorrhages, (2) œdema, (3) external hydrocephalus and (4) exhaustion following a convulsive seizure. These possibilities must be eliminated, if necessary by direct inspection through a trephine hole before a diagnosis of thrombosis of the middle cerebral artery is made. Thrombosis of the middle cerebral artery occasionally occurs and leads to extensive cortical atrophy, which can be demonstrated by ventriculography or encephalography. A paralysis due to a tear of the brachial plexus is an occasional happening and is difficult to diagnose, particularly as paralyses, both of peripheral and central origin, are flaccid in the early stages. A differential diagnosis, however, is possible. In the peripheral type the skin is insensitive and there is no reaction when it is pinched or pricked with a pin, whereas in paralysis of central origin the skin is sensitive, and when a painful stimulus is applied to it, even though the limb itself cannot be moved, some kind of response occurs elsewhere in the body—possibly a change of expression or a sweeping movement of the other arm. Later, when spasticity or wasting has occurred, the

diagnosis is unequivocal. Also, peripheral injuries are often accompanied by evidence of bruising or thickening in the triangles of the neck.

Restlessness is one of the most common features of any injury to the brain. In many cases a patient lurches from one side of the bed to the other as if seeking a more comfortable position, or he pushes and pulls at the bedclothes incessantly Often he attempts to get out of bed in a meaningless kind of way. What the significance of all this activity may be is difficult to know. Probably it is the reaction of the patient to the meningeal pain of a subarachnoid hæmorrhage, associated with bruising of the frontal lobes.

In delirium, movements are incessant, excessive, writhing in character and apparently purposeless. Whether this is the result of pain, hallucinations or delusions is not known, but it is always a grave sign and indicative of severe bruising of the brain, associated with profuse subarachnoid bleeding.

Normal posture and smooth, purposeful co-ordination of movements are indicative of the milder types of cerebral injury and usually mean that the chances of recovery are good.

Aphasia.—Aphasia is an inability, not due to unconsciousness, either to use or to comprehend language in any of its forms

There are two varieties—sensory or receptive, and motor or expressive—and in many cases there is a combination of both these conditions in which one or other of the elements is dominant.

Sensory aphasia was localised by Wernicke to the region of the left supramarginal gyrus in right-handed people, and it is characterised by an inability to understand the spoken or written word

Motor aphasia is loss of power to speak, write or draw, although the messages of writing and speech can be understood Motor aphasia was localised by Broca to the region of the posterior end of the left inferior convolution of the frontal lobe, but there is much evidence to show that a more extensive area of the brain is concerned

Many types of aphasia exist, and formal terms are given to certain types in which a particular modality of language is effected Dysphasia implies that the command of language has been impaired but not entirely lost Agraphia means inability to write, alexia inability to read and apraxia difficulty in knowing how to use a simple machine or object, such as scissors or a pencil.

All degrees and types of aphasia are encountered in acute cerebral trauma, and recognition of this condition is important not only because of its localising value but because it may give the illusion that unconsciousness is deeper than it really is A warning must be given that if an important decision has to be made as regards surgical exploration on the sign of aphasia, it is

imperative to seek from some third person the information as to whether the patient is right-handed. It is unwise to assume that he is right-handed, because in one of my cases, had this precaution not been taken, the wrong side of the skull would have been opened and a large subdural hæmorrhage overlooked.

Motor aphasia is frequently mistaken for confusion, although the differentiation of the two states is not particularly difficult. An aphasic patient has a conscious look about him, and when spoken to some kind of intelligent expression comes into his face, and at best he will obey a complicated request. A confused patient, on the other hand, looks bewildered and can understand only a simple command He ignores anything difficult or complicated.

When a patient comes to a stage of consciousness sufficient to permit recognition of aphasia, he has a very good chance of surviving his injury and most probably will eventually recover his ability to speak with little residual impairment. In two cases only have I known aphasia of any noticeable degree persist, and these were due to extensive depressed fractures which had not been raised by surgical means.

Differentiation between sensory aphasia and severer grades of confusion is impossible. In the phase of cerebral irritation a patient may talk incoherently when stimulated and give the impression to his relatives or to unskilled observers that he has gone out of his mind It is very gratifying to be able to say at an early stage with confidence that this state will probably subside and that the patient will not be mentally impaired. Occasionally, on the sign of aphasia, I have been able to localise a progressive lesion and to centre a surgical exploration correctly for the removal of a surface clot

Aphasia is a most reliable localising sign but its pathological cause is often a matter of conjecture It may be due (1) to contusion or laceration of the areas concerned, (2) to compression by a clot or depressed fragment of bone, (3) to œdema or (4) to thrombosis of a large vessel, and a diagnosis has often to be made through an inspection hole.

Position and Movements of the Eyes.—In concussion a patient's eyes are usually closed. When they are not it is a sign either of approaching consciousness or coma, and a differentiation of these two states is a very simple matter In coma the corneal reflexes are absent or sluggish, whereas in the nearly conscious patient they are extremely active. Also, other tests for determination of depths of unconsciousness are unequivocal.

In those cases when it is necessary to open the eyelids passively the patient will often resist and try, by some kind of purposive

movement, to escape the irritation of light or to avoid the unpleasantness of having his eyelids manipulated. The more purposively the patient resists, the nearer he is to consciousness. For example, a sweep of the hand is indicative of a more complex



FIG 48

A fracture of the lateral wall of the orbit may not only lead to displacement of the eye but to injury of the external rectus muscle or its nerve supply, with resulting diplopia

intellectual process than a mere movement of the head away from the painful stimulus. In spite of resistance a patient's eyelids should be kept open long enough to permit observation of the position of the eyes and of their movements. When movements of the eyes do not occur spontaneously, they may be induced

by moving the patient's head gently from side to side or by twisting his body.

Abnormal positions of the eye and inco-ordinated or restricted movements may be produced by one of the following conditions :—

(i) Hæmorrhages into the orbit.
(ii) Damage to the extra-ocular muscles or destruction of their pulleys by fractures of the orbit.
(iii) Contusion, severance or avulsion of the third, fourth or sixth cranial nerves.
(iv) Injury to the ocular nuclei or their connecting pathways.
(v) Labyrinthine and cerebellar concussion.
(vi) Impairment of function in the visuo-psychic fields.

Displacement of the axis of the eye is due either to extensive extra-orbital hæmorrhages or to deformity of the orbital cavity by fracture of its walls (Fig. 48). Bony damage to the orbit is a common occurrence and is due to the frequency with which patients are thrown on to the face Usually the floor is broken, so that the eye sinks downwards. Upward displacements are rare, but some degree of lateral displacement is often seen.



Fig 49
A third-nerve paralysis due to a fracture involving the sphenoidal fissure

Squints may be complete or incomplete, fixed or changeable. A complete and fixed strabismus is due to damage of an extra-ocular muscle or to contusion, laceration or avulsion of the nerve which supplies it (Fig. 49) An extra-ocular nerve may be damaged in any part of its course, although this usually occurs close to the brain stem or within the orbit.

Skew deviations are often seen and are due to both eyes being pulled away from the axis in which they would normally be resting at any given moment. In other words, a skew deviation is a bilateral strabismus in the vertical plane. Such ocular divergencies are usually associated with inco-ordinate wanderings of the eyes in which both globes wobble spontaneously about the orbit without any reference to each other. These conditions of abnormal posture and movement are due to changes within the brain stem affecting the ocular nuclei or their connecting

pathways. Hæmorrhages into the brain stem in concussion are known to be common, but these rarely destroy the ocular nuclei, because very few patients are left with a permanent strabismus even after a severe head injury. Circulatory disturbances within the brain stem probably account for most skew deviations and inco-ordinations of movement. Also, a vascular basis explains why they are so rapidly changeable in form and degree and why they so often disappear without leaving any residual disability. Occasionally the eyes are fixed in the middle axis and nothing that may be done in the way of stimulation will induce them to move. In these cases the injury involves the posterior longitudinal bundle which is the main co-ordinating tract for the ocular nuclei.

Fine nystagmus or rapid and regular oscillations of the eye are of frequent occurrence and are due to concussion of the labyrinth. Coarse nystagmus is rather less common and is due to contusion of the cerebellum. In this case the oscillatory movements of the eyes have a wider range than in labyrinthine nystagmus, and there is a slow swinging component away from the point of fixation followed by a rapid corrective jerk backwards.

When in the state of confusion a patient will often look at the examiner in a meaningless kind of way, staring vacantly forwards. Also, his sight cannot be directed towards an object or activity which would attract the gaze of a normal person. Occasionally this state is due to apraxia and results from damage to the visuo-psychic centres.

Of all the neurological signs which occur in acute cerebral trauma abnormal postures and movements of the eyes are the most frequent, apart from unconsciousness and restlessness, and indicate that the brain stem is usually affected when a patient is concussed.

The Pupils.—The pupils may be contracted or dilated. Often they are unequal, and rapid alterations in their size are common. Usually they react to light by constriction, but occasionally may dilate in a paradoxical way on stimulation. Such pupillary changes are indicative of an injury to the brain stem and have much the same meaning as alterations in the movements of the eyes. In other words, they point to intrinsic rather than to compressional changes, and although of pathological importance they are of no great surgical significance.

Alternatively, the so-called "Fixed Dilated Pupil of Hutchinson" is a most important sign, both of localising value and pathological inference. In this condition the pupil is fully dilated and it does not contract when a bright light is shone into it or into the opposite eye (Fig. 50). It is necessary to prove that the consensual, as well as the direct, light reflex is absent before

diagnosing Hutchinson's pupil, because in an eye blinded by an intra-ocular hæmorrhage or by severance of its optic nerve the pupil is dilated and does not contract to light, although it does so when the opposite retina is stimulated. A "fixed dilated pupil" is an infallible sign of raised intracranial pressure, and means that a tentorial pressure cone has developed which is compressing the brain stem and stretching the oculo-motor nerve at the point where it is about to enter the wall of the cavernous sinus. Surgically its significance is of paramount importance, for if the uncinate herniation is not relieved actively the patient is



FIG 50
In a fixed dilated pupil the consensual, as well as the direct, light reflex is absent

almost certain to succumb, and, as will be shown later, explorations on both sides of the skull may be necessary in these cases.

Small fixed pupils which remain contracted in spite of changes in the intracranial condition often indicate a hæmorrhage into the pons, and such a diagnosis is more than probable when bilateral pyramidal signs are present and when the temperature rises suddenly and continues to rise

Pulse and Blood Pressure.—In the early stages of primary shock the pulse is rapid and thready and the blood pressure is low, but often when the patient has been put to bed and warmth applied the circulation rapidly improves. When stasis of capillary circulation persists in spite of accepted methods of resuscitation, this is a very grave prognostic sign, and the patient will probably succumb within twelve or, at the most, twenty-four hours. Raised intracranial tension causing medullary compression may produce a compensatory rise in systolic pressure without

improvement of the diastolic pressure, which remains dangerously low. Depth of shock may thus be masked if diastolic pressure is not known.

In states of mild confusion the pulse rate and blood pressure are usually within normal limits, and, given that the mental condition is improving, they are of no particular prognostic or diagnostic value. Furthermore, in those conditions where the patient becomes gradually more drowsy the circulation does not in the early stages deteriorate, as compensations within very wide limits are possible. Therefore, in the presence of other signs of deterioration of the intracranial conditions, a favourable pulse rate and pressure may be misleading.

In states of severe confusion and in semicoma the pulse rate is usually raised to between 90 and 110 beats per minute, whereas the blood pressure, as far as can be judged without comparison with a pre-accident reading, remains within normal limits.[1] Gradual changes in depths of unconsciousness in coma or in semicoma are very difficult to appreciate, and often the first indication is an alteration in pulse pressure and pulse rate A patient may remain unconscious for several days without any apparent variation in his mental condition, and so long as his circulation does not deteriorate there is every reason to hope that he will recover Alternatively, as soon as the pulse rate begins to rise and its volume to fail the whole picture changes and the chances of recovery are poor

A fast bounding pulse is a sign of raised intracranial pressure It is often seen in comatosed patients with stertorous breathing, and indicates embarrassment of medullary circulation It is, of course, a much more favourable sign than a weak thready pulse, as it means that vigorous efforts of compensation are being made to maintain the essential cerebral circulations

Bradycardia or a slow pulse is a condition far more often described than seen, and is in fact a very great rarity in the acute phases of a head injury. In convalescence it occasionally occurs, but is of no particular diagnostic or therapeutic significance On the other hand, it is of very great importance when a patient is unconscious, as under these conditions it means that the brain is being severely compressed and probably by an extradural or subdural hæmorrhage. As a working rule a combination of a slow pulse, below sixty beats per minute, and unconsciousness must always be taken to mean an extradural hæmorrhage until proved otherwise.

In middle meningeal hæmorrhages in which there has been a

[1] Woodhall, B "Acute Cerebral Injuries Analysis of Temperature, Pulse and Respiration Curves" *Arch Surg*, 1936, **33**, 560

latent interval the pulse rate and blood pressure usually show the following changes. While the patient is conscious they are normal; then, as drowsiness develops, the pulse rate increases and blood pressure rises. As unconsciousness deepens, the pressure rises and the pulse rate falls and may become as low as forty beats a minute Occasionally the pulse rate remains high and never falls below normal. Finally, as the cerebral circulation becomes inadequate, the systemic circulation also fails, with the result that the blood pressure falls and the pulse becomes rapid and thready.

Temperature.—When first brought in to the wards from the streets patients are shocked and chilled and consequently their temperatures are subnormal. Later, as heat is applied, body temperature rises, and in coma a patient may very easily be overheated if the temperature of the bed and room in which he is being nursed is not carefully regulated.

In semicoma, apart from environmental influences, a rise of temperature of 1° or 2° F. is common and is due to absorption of extravasated blood. Fluctuation of temperature within these limits in the first few days is usual and is of no particular surgical significance A secondary rise after the temperature has been stabilised for a day or more is often a very serious sign, as it may indicate renewed subarachnoid bleeding or the development of pneumonia or meningitis.

After severe intrinsic injuries of the brain patients often die in hyperthermia The temperature rises as soon as shock has passed, and it continues to do so in spite of cold sponging and may reach as high as 111° F. before the patient succumbs. Any rise of temperature above 101° F., whatever the depth of unconsciousness, is a very grave sign, as it is so often indicative of a severe intrinsic injury to the brain or of a profuse subarachnoid hæmorrhage.

Differences of one or two degrees of temperature between the two sides of the body are occasionally found and are due to interference with the sympathetic nervous system, but they throw no light on the exact site or on the nature of the injury.

Papillœdema.—Papillœdema is so rarely seen in the early phases of concussion that routine retinoscopy is apt to be omitted. This omission is a serious mistake, since swelling of the optic discs, when it does occur, is an equivocal sign of raised intracranial pressure.

When papillœdema develops in the acute phases of cerebral trauma, immediate relief of pressure by intravenous dehydration, spinal drainage, exploration or decompression is indicated, particularly when there are signs of deepening unconsciousness or other evidence of retrogression.

Absence of papilloedema does not mean that intracranial tension is not raised, as drainage of the retina is not impaired until pressures higher than those usually found in cerebral trauma are reached. In acute expanding lesions, such as middle meningeal hæmorrhages, retinal evidence of pressure no doubt would be more often seen if the patient did not succumb rapidly without surgical intervention.

In my own experience papilloedema has never occurred before the third day, and only in cases of obstructive hydrocephalus and generalised œdema.

Respiration.—Normal respiration is a good prognostic sign, as it means that the brain has not received a severe intrinsic injury and is not being seriously compressed by a surface hæmorrhage

Increased rate and depth of breathing which occur in restlessness can be explained by physiological adjustments rather than by pathological processes

Stertor is usually a sign of impending death. It is seen in the early stages of coma when loss of muscle tone allows the jaw and tongue to fall backwards to impede respiration and occurs just before respiration is about to fail.

Deviation from normal rhythm indicates failure of medullary circulation, and the patient will almost certainly die if the nature of the lesion is such that it cannot be relieved surgically.

The autonomic or respiratory centre proper is a complex structure and is thought to be situated in the pons and upper part of the medulla. Extremely different types of breathing develop when transections of the brain stem are made from above downwards. The upper part of the centre is concerned with normal or pneumotaxic breathing, the middle with apneustic and the lower with gasping breathing Apneustic breathing occurs when the transection is made just below the inferior colliculi and the rhythm is as follows First the patient takes a slow, deep inspiration which he holds for one or two minutes This is followed by a sudden relaxation of the inspiratory muscles and the air is suddenly expressed from the chest After a few normal breaths the apneustic cycle is repeated

In gasping breathing, inspiration and expiration begin and end suddenly and are followed by a pause. This type of breathing occurs when the transection is made at the level of the striæ acoustici. A transection at the calamus scriptorius causes cessation of respiration.

Breathing in middle meningeal hæmorrhages and other expanding lesions first becomes deep and fast, then deep and slow, and finally irregular. Changes in irregularity throw light on the functions of the complicated respiratory centre, and it is often

educational to sit and listen to alterations in breathing in a comatosed patient as the various centres fail from above downwards.

Intracranial Pressure.—Apart from direct inspection of the brain through surgical exposures, spinal and ventricular manometry are the only reliable methods of measuring intracranial tension, since deductions from symptoms and clinical signs are apt to be misleading.

Measurements are made in millimetres of cerebrospinal fluid; normal pressure in the lateral position registering between 50 mm. and 150 mm.

INTRACRANIAL PRESSURE IN TWO HUNDRED CASES OF TRAUMATIC UNCONSCIOUSNESS

Pressure of C S F	Number of Cases
Over 300 mm	20
Between 200 to 300 mm	146
Normal	30
Decrease	4

All these readings were taken within twenty-four hours of the injury

In the above series the highest pressures were found in restless patients whose cerebrospinal fluid was heavily stained with blood. On one occasion over 500 mm. was registered, and in this case the bloodstained cerebrospinal fluid shot out of the top of the manometer as if the spinal needle had been introduced into the abdominal aorta. Low normal or subnormal pressures occur particularly in shock · in old people with poorly nourished bodies; in the later stages of coma; and in those cases where there has been a loss of cerebrospinal fluid from the nose, ears or into the subgaleal space.

Usually intracranial pressure becomes stabilised within normal limits after one or two spinal drainages in which sufficient fluid is withdrawn to bring the pressure to 50 mm.

From spinal manometry alone no precise deduction can be made regarding the pathological state within the cranium, since increased pressure may be due to (1) extravasated blood, (2) increased volume of blood within the cranial vessels, (3) cerebral œdema, (4) hydrocephalus.

Furthermore, depth of unconsciousness cannot be judged by spinal manometry alone.

Russell[1] found that on twelve occasions when the pressure

[1] Russell, W R "Discussion on Intracranial Pressure its Clinical and Pathological Importance" *Proc Roy Soc Med*, 1934*b*, **27**, 832

was above 200 mm the patients were fully conscious, whereas in seven stuporous patients the pressure was below 200 mm. Also, in large extradural hæmorrhages subnormal tensions may occur, as Jefferson has shown. In my experience a low or subnormal intracranial tension in comatose patients is a bad prognostic sign and probably indicates that the cerebral circulation is failing. Alternatively, when intracranial pressure is high a stuporous patient's life is still in the balance, and efforts to save it will often be rewarded by gratifying results.

Changes of pressure in a conscious patient are of very little significance as regards life and death because the underlying cause, whatever this may be, is so often amenable to treatment

Although intracranial pressures above 500 mm. are often found in cases of tumours of the brain in which consciousness is not impaired, this does not mean unconsciousness cannot be produced by the smaller rises of tension in cerebral trauma. It is the speed at which the pressure develops rather than its magnitude which is important Rapidly expanding lesions do not give time for compensation of cerebral circulation to take place as is the case in those which develop more slowly

In one case of an acute subdural hæmorrhage which was unassociated with intrinsic injury of the brain, I was able by spinal manometry to demonstrate a gradual rise in intracranial pressure up to 300 mm., at which point the patient died. (Decompression was not done in this case because the patient succumbed within half an hour of admission to hospital, whilst preparations for operation were being made)

If, as is generally believed, increased intracranial pressure, as distinct from the pressure of a large clot, has a deleterious effect on recovery of local or diffuse contusions of the brain, treatment ought to be designed to keep the intracranial pressure within normal limits. Whether this is done by decompression, lumbar and ventricular drainage or by intravenous dehydration, depends on the cause of the rise in pressure. Intravenous dehydration, for example, can only be of value in cases of cerebral œdema, in other cases it is harmful

The presence of blood in the cerebrospinal fluid, of course, establishes the diagnosis of subarachnoid hæmorrhage and, as stated previously, will be found in 75 per cent of cases of severe concussion It does not, however, localise the lesion or distinguish between venous or arterial bleeding. The amount of blood present is of prognostic value only within wide limits According to Russell, patients are always comatose when more than 100,000 red blood cells are found in the cerebrospinal fluid

In my own experience profuse bleeding associated with

unconsciousness, has usually ended fatally, and the greatest quantities have been found in gunshot injuries. In one case in which a bullet passed through the whole head the blood clotted in the test-tube as it was withdrawn through the spinal needle. Raised intracranial pressure in the absence of subarachnoid bleeding is indicative of œdema or of hydrocephalus.

Ventriculography [1-3] (Fig. 51).—Ventriculography is the replacement of ventricular fluid by air or oxygen in order to

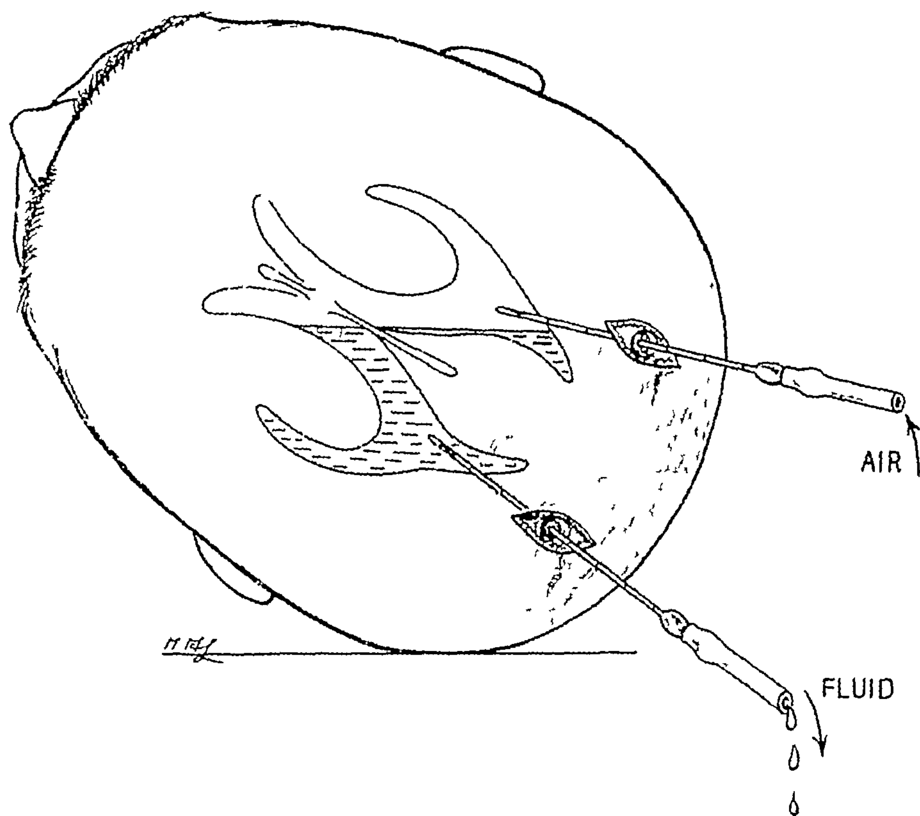


FIG. 51

Ventricular puncture and replacement of ventricular fluid by air. Note the position of the head to ensure complete replacement. The scalp incisions are held open and bleeding from them is controlled by small (mastoid) self-retaining retractors. The incisions are usually placed somewhat farther back than the drawing indicates. (*Norman Dott.*)

produce a silhouette of the ventricular system which is demonstrable by X-rays. It is performed by passing a brain cannula into the ventricles through a hole bored in the skull 9 cm. above

[1] Dandy, W. E. "Ventriculography following the Injection of Air into the Cerebral Ventricles." *Ann. Surg.*, 1918, **68**, 5.
[2] Childe, A. E., and McConnell, L. H. "Pneumographic Localisation of Tumors of the Brain." "(i) Tumors of the Lobes of the Cerebrum, (ii) Tumors involving the Basal Ganglia, Lateral Ventricles, Brain Stem and Cerebellum." *Arch. Neur. Psych.*, 1937, **37**, 33-55 and 56-67.
[3] Shanks, S. C., Kersley, P., and Twining, E. W. "A Textbook of X-ray Diagnosis." Lewis & Co. London, 1938.

the external occipital protuberance and 3 cm. from the middle line, the cannula being so guided that it aims at the pupil of the same side. The ventricle is found at a depth of about 5 cm. from the surface. Cerebrospinal fluid is allowed to drain into a graduated test-tube or is withdrawn slowly into a syringe in amounts of 5 c.c. and replaced by injections of similar amounts of air. It is preferable to tap both lateral ventricles, and in this case 5 to 10 c.c of air on each side is all that is necessary to give a satisfactory



FIG 52

A ventriculogram showing displacement of the left ventricle to the left side In this case it was not possible to tap the right ventricle, presumably because of displacement

picture. By means of ventriculography, enlargement, collapse, deformity or displacement of any part of the ventricular system can be clearly visualised (Fig. 52).

Encephalography.—Encephalography is the replacement of cerebrospinal fluid by oxygen or air through a spinal puncture performed either at the level of the cisterna magna (cerebello-medullary) or at the lumbar theca In this investigation the patient is put on the operating table and a lumbar puncture performed in the left lateral position Cisternal punctures are better avoided in restless patients, lest an uncontrolled movement cause the needle to prick the medulla A block or suitable support is then fixed behind the

patient's thigh or buttocks and the table tilted at least 45°, with the head uppermost. Ten cubic centimetres of fluid are then withdrawn and replaced by an equal amount of whichever gas is chosen for injection. At least 45 c.c. of air must be introduced and more if detailed pictures are considered necessary. The advantage of oxygen over air is that it is more rapidly absorbed and postoperative discomfort is less severe. Long experience has shown that air taken from the room and not subjected to any special processes of sterilisation may be injected without causing meningitis.

The radiographic advantage of encephalography and ventriculography *is that the gyri and cisterns*, as well as the ventricles, are outlined.

One of the great advantages in diagnosis of tumours of the brain is that ventriculography can be used freely with reasonable safety, and is always permissible when localisation is in doubt.

On the other hand, in acute cerebral trauma its use is restricted by its attendant dangers and is indicated only on special occasions.

The main indications for ventriculography are :—

(i) When unconsciousness has lasted a whole day at the same depth and is then followed by retrogression.

(ii) When depth of unconsciousness has not changed after thirty-six hours.

(iii) Failure to expose a suspected subdural hæmatoma through inspection holes bored in the skull.

The main indications for encephalography are :—

(i) Lack of localising signs when a large clot or any other form of compression is suspected.

(ii) When definite localising signs, such as hemiplegia or aphasia, are present.

Ventriculography is a safer procedure than encephalography for the following reasons :—

(i) Less air or oxygen needs to be injected to give useful pictures.

(ii) The gas can be removed easily at the end of the examination.

(iii) There is less likelihood of a pressure cone being precipitated.

Properly used, ventriculography is a means of saving life, and at the moment there is a growing opinion in authoritative circles that ventriculography should be used more frequently than has hitherto been customary, particularly since drainage of the

cerebrospinal fluid through a ventricular tap is the best method of maintaining intracranial pressure within normal limits.

Electro-encephalography.—When a man's eyes are closed and his mind is at rest, oscillations of electrical potential occur on the surface of the head. These consist of a series of waves with a frequency of ten per second and an amplitude of 0 05 to 0 1 millivolts.

Berger [1] first described these oscillations in 1929, and they are now known as the Berger Rhythm. According to Adrian and Matthews,[2] they arise in the occipital cortex and are neutralised when other parts of the brain become active Waves of other shapes and frequencies have been described, and in 1936 Grey Walter,[3] working at the Maida Vale Hospital for Nervous Diseases, put these discoveries to direct use, as a result of which the electro-encephalogram is becoming a routine part of neurological diagnosis Pathological states alter the electrical activities of the part of the brain concerned and, after suitable amplification, such alteration can be measured and located by correct placing of electrodes

The apparatus is not simple, and a great deal of experience is necessary to interpret the records An important advantage of this method over air encephalography, however, is that the patient is put to little inconvenience and no harm is likely to be done All that is necessary from his point of view is that his scalp and hair should be clean

A cerebral tumour is electrically dead compared with normal cortex and, when on the surface of the brain, can be located with some certainty In head injuries, however, the problem becomes rather more difficult because the injury is diffuse. In my own opinion the primary use of the electro-encephalogram is in the detection of large surface clots. Later, should complications occur, it is of value in localising an epileptogenic focus and in throwing light on the nature of the neuronal discharge.

INDICATIONS FOR SURGICAL TREATMENT

Even after the most careful examinations it is not always possible to interpret the many clinical signs observed in terms of pathological lesions with that precise accuracy necessary to guide successful treatment It is therefore proposed to review the diagnostic problem from a different angle even at the risk of repetition From the clinical standpoint all cerebral injuries may be classified into three main groups according to the degree of

[1] Berger, H *Arch f Psych*, 1929, **87**, 527
[2] Adrian, E D, and Matthews, B H C *Jour Physiol*, 1934*a*, **81**, 440
[3] Grey Walter, W "Electro encephalography in Cases of Cerebral Tumour" *Proc. Roy Soc Med*, 1936-37, **30**, 579

unconsciousness when the patient is first seen, and according to the way in which unconsciousness developed.

Group I.—Group I, which is always the largest in any clinical series, includes those patients who are conscious or who are rapidly approaching consciousness when first seen

Although opportunities to observe the earliest phases of concussion rarely occur it is important to be acquainted with the sequence of events in this stage, as it elucidates many of the problems which may arise later and gives a guide to the correct form of treatment

In a typical case a man receives an injury to the head and either falls or remains on the ground completely unconscious In this stage there is complete paralysis of function which, as Symonds has stated, may extend for a moment to the vital centres. Then after a few moments recovery begins in order of sequence from the lowest to the highest neurological levels. First, the pulse and respiration return, but the patient remains in a state of coma with flaccid muscles. Possibly this is the phase of medullary control. Soon the muscles regain their tone and the reflexes return, which probably means that the upper part of the brain stem is recovering its function. Then, as the cerebrum recovers, consciousness returns The patient moves a limb, then makes a purposive movement, usually with his hands, and soon tries to sit up. At this stage he becomes restless, attempts to speak, but is confused and often becomes resistive and even violent Before final recovery he reaches the stage of automatism, which is a condition of extreme medico-legal importance, for although he behaves naturally and is apparently in full control of his faculties, he is not really responsible for his actions He can stand up, can converse logically and answer questions correctly. The danger is that, unknowingly, he may make statements which are untrue He may start up his car and drive carelessly or he may behave dangerously in some other way to himself or others without later being aware of what has happened Statements taken immediately after concussion should be accepted with a good deal of circumspection In the less severe cases a patient may be dazed for a moment only. Things go black before his eyes, but before he falls or loses his balance he regains his senses and goes on as usual.

The state of slight concussion in games is very well known and many of us have experienced it. A player receives a knock on the head and is dazed for a few moments but does not leave the field He continues to play, but automatically and badly. He misjudges the ball in a way he would not do in the ordinary course of events, and does not show that initiative or sense of anticipation which has gained him his position in the team.

When a conscious patient is admitted to hospital giving a history of concussion, no particular medical or surgical treatment is necessary if there are no residual signs of injury to the nervous system, and the only decision to be made is whether he should be detained or not.

This decision is important, because a patient who has apparently made a complete recovery from concussion may suddenly develop a fatal secondary compression. It is safer, therefore, that all patients who have received an injury to the head of sufficient degree to bring them to hospital should be kept under observation for a few hours, and those who have been definitely unconscious should be detained for at least one night. If this is done it can be claimed that reasonable care has been taken whatever may happen later in the way of complications. An X-ray of the skull is also advisable because of the importance the public attaches to a fracture. Finally, on discharge from hospital, a message should be sent to the patient's general practitioner requesting him to send the patient back to hospital if severe headache or drowsiness develops.

When local brain damage has occurred a patient should be admitted to hospital at once for full neurological investigation. Signs and symptoms of local injury, as Symonds has pointed out, are associated most frequently with those of generalised brain injury, but they may occur with little or no disturbance of consciousness. Also, they vary in kind and number according to the site and nature of the injury. Cranial nerves may be avulsed, special centres may be contused and important blood vessels ruptured or thrombosed.

The results of injury to special parts of the skull and brain will be discussed in a succeeding chapter, but for the sake of continuity the subject will have to be introduced here.

Blows on the top of the head may cause paralysis of both legs. Recently I have seen three such cases, and on each occasion the patient was badly dazed by the blow but soon regained consciousness and was co-operative at the time he reached hospital. Both legs were spastic, the knee jerks were increased and the plantar response was extensor. Sensory changes were present, although these were subjective rather than objective On testing, no modality of sensation was definitely lost, the feeling being that a lighter stimulus was being applied than to the normal parts of the body. In other words, the skin felt as though it were being touched through the clothes. Gordon Holmes [1] described this syndrome in detail during the last war and suggested that some of

[1] Holmes, Gordon, and Sargent, P "Injuries of the Superior Longitudinal Sinus" *Brit Med Jour*, 1915, 2, 493

these cases are due to occlusion of the superior longitudinal sinus, which interferes with drainage of the motor cortices, thus explaining the bilateral signs.

Hemiplegia and monoplegia may also follow an injury which causes no more than momentary loss of consciousness and, as in the case of unconsciousness, may occur at the moment of violence or develop after an interval.

Immediate paralysis may be caused by one or a combination of the following conditions: (1) neural shock, (2) exhaustion following epileptic seizures, (3) compression by depressed bony fragments, (4) contusion and (5) laceration. Bony compression may readily be confirmed or eliminated by radiography. In the other cases diagnosis depends largely on the subsequent events. Recovery from neural shock and epileptic exhaustion is usually rapid and often complete within twenty-four hours. Paralysis due to contusion usually begins to improve within a few days and full recovery of function is the rule. Prognosis is serious in lacerations. Fortunately this is a very rare condition in non-fatal closed injuries and is invariably associated with a closed depressed fracture.

Interval paralysis may be due to (1) compression by a surface hæmorrhage, (2) compression by an external hydrocephalus, (3) œdema, (4) arterial occlusion and (5) spreading venous thrombosis. Prognosis as regards recovery of motor power is very much worse in delayed than in immediate paralysis, because intrinsic lesions are much more common than surface compressions. The onset of paralysis is often progressive, save in the case of intracerebral hæmorrhage which is ushered in as an acute cerebral crisis. Delayed post-traumatic intracerebral hæmorrhages often occur in middle-aged people with normal blood pressures. Typically, the episode occurs in the patient's bedroom. He feels giddy, attempts to get out of bed and falls. When he is lifted it is found that he is paralysed. Full consciousness soon returns. Spinal pressure is not appreciably raised, and neither blood nor abnormal chemical constituents are found in the cerebrospinal fluid. Encephalography, however, shows a filling defect in the lateral ventricle on the affected side. Considerable recovery may be expected in these cases, but a certain amount of clumsy movement and impairment of the finer movements of the fingers will almost certainly remain. In secondary paralysis, diagnosis of the underlying cause should always be made by inspection through trephine holes or preferably by air replacement.

Group II.—Group II consists of those patients who are unconscious when first seen, but who have been conscious during some period after the accident. This is a very small group, but it is important from the surgical point of view. Whether the

patient was unconscious before the latent interval is of prognostic importance, since absence of early unconsciousness means absence of intrinsic brain damage.

Meningeal Hæmorrhages—The latent interval in middle meningeal hæmorrhages, as shown in the previous chapter, is usually a number of hours only, although it may be as long as a week or more The first symptom is increasing headache, and this must never be treated lightly when a history of an injury, however slight, has been given. Giddiness, mental confusion or drowsiness must be regarded as pathognomonic and not merely as suspicious signs of extradural compression. As the hæmorrhage increases, drowsiness or confusion changes to unconsciousness and signs of pyramidal impairment develop on the opposite side of the body. At first the limbs become weak and spastic. Convulsive twitchings may occur at any stage either early or late. The reflexes are typical of those of an upper motor neurone lesion; the knee jerk is increased, the abdominal reflexes on the same side are diminished or absent, and there is an extensor plantar response Respiration, at first fast and deep, slows and then becomes irregular The pulse in a fully developed case is slow, *i e.*, below sixty beats a minute, and the blood pressure is high. A fixed dilated pupil may or may not be present and in any case is usually a late sign.

When pyramidal signs are confined to one side of the body, lateralisation of the hæmorrhage is easy, but if the clot is not evacuated at this stage bilateral motor signs rapidly appear and diagnosis is much more difficult Often a patient is first seen when bilateral signs are well developed In these cases there may be (1) bilateral spasticity, (2) spasticity on one side and flaccidity on the other or (3) bilateral flaccidity These possible combinations are explained by the way the clot compresses the brain First the cortex on the side of the hæmorrhage is irritated, and this may lead to convulsive seizures in the contralateral limbs Then venous congestion develops at the site of compression and causes a spastic type of paralysis. Later the cortex becomes ischæmic and a spastic changes to a flaccid paralysis. Finally, as the clot expands, a similar sequel of phenomena occurs on the opposite side of the brain and the combination of clinical signs found at any time depend on the phase of compression

In a combination of flaccid and spastic paralysis the clot, therefore, is on the side of the brain opposite to the flaccid paralysis. In those cases where there is no difference between the two sides of the body, lateralisation of the clot on physical signs alone is impossible, but the diagnosis may be made if the sequence of events is known.

Subdural Hæmorrhages.—In subdural hæmorrhages the latent interval is usually longer than in middle meningeal hæmorrhages, and signs of compression may not develop for days or even years. Repeated or increasingly severe headaches associated with mental changes, such as drowsiness or apathy, are the first indications of compression. Later signs and symptoms are extremely variable. Periods of unconsciousness may come and go, papillœdema may or may not be present and localising signs are often absent. The important fact in the diagnosis of a chronic subdural hæmatoma is that a relatively slow-developing cerebral crisis has occurred at some time after a complete or a partial recovery from an injury. In those cases when it is not possible to localise a clot on neurological evidence, such as a hemiplegia or a facial paralysis, it must be found by inspection through exploratory trephine holes. Usually it is discovered high over the parietal lobes or just above the Sylvian points.

When a large blood clot cannot be found in spite of exploration, encephalography or ventriculography is indicated, but if the facilities for this procedure are not ideal it is best to perform a right-sided subtemporal decompression and then to seek expert aid.

Group III.—Group III consists of those patients who are unconscious and have not been conscious at any time since the accident.

Cases of Favourable Prognosis.—In this sub-group the patients are not deeply unconscious. At most they are confused or drowsy, and although they may resent interference or be unable to co-operate in a complicated examination, they will obey a simple command or imitate a simple gesture. Restlessness may be present, but this is not incessant or of a lurching character and often the patient will lie in a perfectly natural position for long periods. Occasionally he will attempt to get out of bed in a purposeless kind of way or in an attempt to empty his bladder. All the primitive reflexes, such as swallowing, are present and, apart from the mental changes, there are no obvious abnormal neurological signs. Pulse, respiration and blood pressure are normal.

Patients suffering from minor neuronal injury or minor contusion are conscious and out of danger usually within twenty-four hours of the receipt of their injury.

Cases of Grave Prognosis.—Many of the cases which are obviously going to die are moribund from the beginning and make no kind of improvement despite all the usual methods of resuscitation. Unconsciousness is so deep that no kind of response can be elicited even by very painful stimuli. The corneal reflexes and tendon jerks are absent, the muscles are flaccid, the jaw is

fallen and, in the worst cases, swallowing is absent. The pulse is fast and feeble, but may for a short period be bounding before it begins to fail Respiration may be stertorous at the start, but soon becomes irregular and later feeble. Often the skin is wet with perspiration. Within an hour or so the temperature begins to rise, signs of basal pneumonia develop and the patient dies in hyperthermia within twelve or twenty-four hours.

Cases of Doubtful Prognosis.—This is the most important group from the diagnostic and therapeutic points of view and the one in which detailed and repeated examinations must be made if logical conclusions are to be drawn The patients are either in coma or semicoma and there are neurological signs referable to damage to the motor system, but there is nothing definite to point to a focal or compressive lesion. In the first twelve hours, therefore, in these cases it is usually necessary only to treat shock and to apply surgical first principles, as will be described later. At the end of this period some cases will obviously be improving, others will be moribund and there will be some in which no apparent recovery or retrogression has taken place. It is this latter group which engages chief attention From the twelve-hour period onwards a knowledge of the intracranial pressure, as measured by spinal manometry, is necessary, since it is believed that the optimum conditions for recovery of damaged cerebral tissue are produced by maintenance of the intracranial pressure within normal limits. Also, repeated examinations are essential so that the development of localising neurological signs or changes in depth of consciousness are observed at the earliest possible moment.

Let us start at the stage when there is mental and physical paralysis. One of the earliest signs of recovery is return of normal tone in the flaccid muscles, and this can be most easily observed by a return of facial expression. Later there may be a spontaneous movement of a limb or there may be an active change from one position to another Reactions to painful stimuli appear as the patient recovers, and the first groan is the first sign of recovery of function of the speech centre With further improvement, restlessness develops and the patient incessantly changes his position. Whether this is the result of physical pain or of a state of subconscious mental anxiety is not known, but probably both factors play an important part

Irritability follows restlessness and may be expressed either by word or deed The patient will often violently resist, by purposive movements, any kind of handling, however gentle, and will knock away a cup or spoon as an attempt is made to feed him. Later he confines his objections to the spoken word He is rude,

impatient, intolerant and lacks all sense of deference, as if there had been a complete break-up of his personality. At times his behaviour may be indecent.

Then comes the stage of mental confusion. The patient begins to waken and wonder what has happened. Although he is able to co-operate to some extent, he is unable to engage in sustained rational conversation At one moment he will recognise his relatives and make some intelligible statement, whereas at the next he will show no interest in them and begin to mutter incoherently or irrelevantly He may sometimes lose emotional control and cry or laugh with equal facility. The most common feature is that he does not realise the gravity or the meaning of his condition and is apt to pick at a wound or try and break down a splint controlling a fracture. Insight for a time is in abeyance and confabulation is common Finally he comes within the limits of intellectual normality, and although mentally sluggish he realises he has received an injury and can assess its importance for both himself and his family His facial expression changes from one of indifference to one of concern and he begins to complain of his aches and pains.

In favourable cases respiration, temperature and the circulation remain within fairly normal limits, any divergence from the normal, even though the mental processes are clearing, is serious, as it may indicate meningitis or pneumonia.

If at any time after twelve hours the patient becomes more deeply unconscious and the circulation fails, a decision has to be made as to whether or not this deterioration is due to the development of secondary phenomena which can be treated by cranial exploration or by decompression of the brain.

This decision is difficult, and it would be misleading to give the impression that subtemporal decompression plays a large part in treatment; it does not. It plays a small though important part, and its correct practice marks the difference between those who are and those who are not experienced in treating acute injuries to the brain.

Neuro-surgeons are not agreed on the question of surgical exploration in closed head injuries; while some discountenance it, others advocate frequent explorations and decompressions through large osteoplastic flaps. The following suggestions are based on my personal experience.

INDICATIONS FOR SUBTEMPORAL DECOMPRESSION

1. Retrogression, following a Period of Improvement which cannot be controlled by Spinal Drainage or by Intravenous

Dehydration.—In this type of case the general condition of the patient progressively improves in a way that cannot be explained by recovery from shock. Unconsciousness becomes less deep, the muscles regain their tone, primitive reflexes become firmly established and abnormal reflex responses disappear. Pulse, respiration and blood pressure remain within, or approaching, normal limits. Usually before these observations can be established a period of at least twelve hours is necessary. Then, for some reason which is not very obvious, the patient's condition begins to deteriorate and, in particular, the depth of unconsciousness increases. Reliable localising signs are rarely found

When there are no localising or lateralising signs, a right-sided decompression should be made. Also, two inspection holes should be sunk on the left side—one at the Sylvian point and one over the parietal eminence in order to eliminate a possible extradural or subdural hæmorrhage. Such conditions, when present on the right side, will be uncovered by the decompression. On those occasions when a large surface hæmorrhage is found, it is not enough merely to remove the clot without decompressing the brain, as the absence of a latent interval means that the brain has received intrinsic damage and probably will become œdematous if it is not already in this state

2. Delayed Decerebrate Rigidity.—When a tentorial herniation develops within twelve hours it usually means that the brain has received a severe intrinsic injury, and very little benefit is to be gained by subtemporal decompression. On the other hand, when decerebrate rigidity develops after twenty-four hours, the causative herniation can often be relieved successfully by surgical decompression A fixed dilated pupil may be present to lateralise the hernia, but when this is absent a bilateral exposure has to be made. At the time of the operation, swelling of the brain usually prevents anything more than a simple opening of the dura, but if the temporal lobe can be lifted without laceration or bruising of its cortex, the hernia may be lifted out of the hiatus tentorii or the tentorium may be split from within outwards

It is in this type of case that the most spectacular results can be obtained.

3. A Fixed Dilated Pupil.—A fixed dilated pupil, even in the absence of decerebrate rigidity, indicates a raised intracranial pressure of such degree that it will lead to a fatal outcome if not relieved If after twelve hours—and it is impossible to judge in a shorter period than this—the patient is not improving and his state is serious, then a decompression ought to be made on the side of the affected pupil

4. Prolonged Unconsciousness associated with Persistently High Cerebrospinal Fluid Pressure.—After thirty-six hours, if a patient has shown no sign of recovery of consciousness and the cerebrospinal fluid returns to a supranormal figure, in spite of repeated spinal drainage or of intravenous dehydration, it is advisable to perform a right-sided subtemporal decompression.

INSPECTION HOLES

In the last few years I have been making frequent use of small inspection holes cut in the skull to establish a diagnosis in doubtful or difficult cases. These holes are placed at points determined by neurological signs, and are made with a trephine so that the disc of bone may be replaced if nothing abnormal from the surgical point of view is found.

McConnell of Dublin has also used this method extensively, and on many occasions he has found a subdural collection of fluid the drainage of which has led to obvious and rapid relief of symptoms

His routine in unconscious patients is to place a hole 1½ to 2 in above the external auditory meatus so that it can be incorporated in a subtemporal decompression if this is found necessary. In those cases where surface hæmorrhage is suspected but cannot be established on clinical data, an inspection hole will save many hours of unnecessary anxiety from the surgeon's point of view, and particularly as it is a procedure that can be carried out rapidly without doing any harm to the patient even if a negative exploration is the result

CHAPTER IV

TREATMENT OF CLOSED INJURIES OF THE HEAD AND SURGICAL TECHNIQUE

FIRST AID and Treatment.—First-aid treatment on the roads is rarely the concern of medical men, since the injured rapidly come under the care of ambulance services and can, under normal circumstances, be removed to hospital within one hour. When a decision regarding transport has to be made on those occasions when a tibia or femur has been broken, or when there is the slightest suspicion of a fractured spine, it is wiser to make the injured person comfortable and warm by covering him with coats and to wait for an ambulance rather than to send him to hospital huddled in the back seat of a private motor car.

Closed head injuries travel well in modern ambulances and may safely be sent long distances for treatment. If distance, therefore, is the only consideration, it is much wiser to transfer a patient directly to a hospital which is specially equipped for treatment of cerebral trauma than to an institution which is not fully conversant with this work. During transport a free airway must be maintained to prevent congestion, and for this reason a comatose patient should be turned on to his side and not allowed to lie on his back, in order to prevent his tongue from falling backwards to impede respiration. This position also allows saliva to trickle outwards which otherwise might be aspirated into the lungs. A small dose of morphia ($\frac{1}{4}$ gr.) may be given when a patient cannot be controlled by gentle restraint, but drugs are better withheld.

It is essential that the surgeon who is finally to take charge of the case should receive sufficient clinical data of the patient's early neurological state to enable him to proceed with treatment in a scientific manner. He must know, for example, whether the patient has been conscious at any time after the accident and, if not, how deeply unconscious he was when first seen. Also, if neurological observations have been made, and this is always desirable when conditions permit, as many details as possible should be passed on and records of drugs administered stated.

NURSING

The surgeon in charge of a Unit concerned in the treatment of cerebral trauma should personally instruct his staff in the details of nursing, and this responsible task should not be delegated to some impersonal lecturer. His Ward Sister should be made responsible for the recording of pulse rate, respiration rate and rhythm, temperature, amount of fluids taken and, under special conditions, for blood-pressure readings and changes in depth of unconsciousness. Records are best kept in the form of a graph on the same chart and preferably in different inks, so as to make them readily distinguishable and easily comparable. Observations in the first twenty-four hours should be made at half-hourly intervals and more frequently when active surgical measures are considered imminent. When a patient is obviously improving, hourly records are sufficient. These are the minimum requirements and are possible in the busiest wards As the staff becomes more highly trained, details, particularly about the state of unconsciousness, will become elaborated. It is by these means only that a continuous clinical picture can be obtained, and this is necessary if a patient is to receive the best possible treatment. Time spent in the training of the nursing staff will be amply rewarded.

Restlessness.—As soon as a patient reaches the restless phase his mind is open to suggestion, when a kind or encouraging word will often be of the greatest help, although at the time it may appear useless. Therefore, before anything is done for a restless patient he should be reassured that it is for his good At times it is surprising how far he can be influenced by the spoken word.

As restlessness is always a trying problem to nurses and attendants without special neurological experience, it is advantageous to stimulate their interest and to gain their co-operation by explaining to them the nature of the underlying disturbance If they realise that a subarachnoid hæmorrhage is a form of meningitis, they will handle a patient gently and refrain from making any forcible movement of the limbs or neck which is likely to cause further pain.

Since restlessness is, to some extent, the result of overaction of the subcortical centres as these are released from the restraining influences of the higher centres, it is wrong to attempt to control movements by forcible restraint, as this will merely act as a further stimulus and the patient will continue to resist until he has exhausted himself By raising and padding the bed sides and by having someone constantly in attendance to prevent a patient from getting out of bed, he may be allowed considerable freedom

of movement without injuring himself. Moreover, there is no particular therapeutic merit in the old established habit of keeping the head low. In fact there are disadvantages, particularly if stretching of the neck muscles causes discomfort and thereby increases restlessness. Drugs in the early stages should be given sparingly, and particularly when prognosis is in the balance. Also, it is safer to repeat small amounts than to give a stiff initial dose.

A routine which I have found useful and safe is as follows. First, soluble luminal (3 gr.) is given intravenously, followed by chloral hydrate (10 gr.) and sodium bromide (10 gr.) by mouth. When a patient will not swallow, double doses of chloral and bromide are given per rectum. The chloral and bromide may be repeated in two hours and afterwards at lengthening intervals until reasonable rest is procured. Paraldehyde is also an excellent drug, and 4 drachms per rectum may be given instead of the chloral and bromide. Morphia is rarely needed, but $\frac{1}{4}$ gr. in combination with luminal will often give a complete night's rest. The principle to be observed in the administration of drugs is to give the minimal amount which will give the desired effect. To give more is dangerous and to give less is useless. Constant medical supervision of drug administration is therefore essential, and should never be allocated to the discretion of a junior member of the staff.

Feeding.—Patients are usually able and willing to swallow. When they cannot do so they must be fed through a tube passed through the nose into the stomach. This tube is fixed to the cheek by adhesive plaster.

Glucose in water is the only drink which need be given in the first twenty-four hours, and the quantity is regulated by the amount of dehydration considered necessary to avoid cerebral œdema. It is obviously illogical to withdraw cerebrospinal fluid by spinal drainage or by intravenous therapy if unlimited quantities of water are simultaneously given by mouth. Theoretically, when dehydration is considered necessary, fluids should be entirely withheld, but under these conditions a patient rapidly becomes toxic, his mouth becomes dried, his tongue furred and he soon looks worse than before treatment started. Therefore in an attempt to relieve possible swelling of the brain the general bodily needs must not be overlooked, and 1-oz. drinks every hour are not only beneficial but also essential. This is the minimum quantity of fluid that should be given in the first twenty-four hours. On the second day larger quantities of fluid may be given according to the state of the patient. When unconsciousness is deep and cerebrospinal fluid pressure high, it should be limited to 2 pints. Less should never be given. When consciousness is

obviously returning, 3 pints may be given if the patient will swallow them. Milk drinks should be given alternately with glucose water.

On the third day it is essential to introduce some kind of protein-containing food, otherwise a patient will start to live on his own tissues and will die from exhaustion. Junket, egg custard, milk puddings and meat jellies will be found useful for this purpose. Fixed rules for feeding, of course, cannot be laid down, and diet must be adapted to the individual needs of each case. More fluid, for example, will have to be given to a patient who is perspiring or passing water freely than to one with dry skin and inactive kidneys. To supply the bodily needs, about 5 oz of milk or its equivalent is necessary every two hours during the day and every four hours during the night

The Bowels.—There is a serious risk of incontinence in giving aperients to an unconscious or semiconscious patient, and obviously this is a very distressing condition when associated with restlessness Whenever it does occur it should be corrected at once by the administration of a mixture containing bismuth and opium Magnesium sulphate by mouth is often advocated in amounts large enough to produce a watery stool, but it is better, in my opinion, to empty the bowel by enemata and to use other methods for dehydration. Cathartics such as calomel should never be given. At best they cause discomfort and often produce colicky pains with resulting restlessness.

The Bladder —Incontinence is not uncommon and necessitates constant changing of the bed linen if a patient is not to become uncomfortable or his skin broken. Retention is usually due to apraxia; that is, a patient is unable to empty his bladder because he does not know what to do In these cases catheterisation is necessary, otherwise the discomfort of a distended bladder will make him restless or compel him to get out of bed in an effort to relieve himself. When a patient is approaching consciousness a bottle or a bed-pan, according to sex, should be placed in a suitable position and the patient requested to pass water. When this device proves successful it should be repeated at three-hourly intervals. To keep incontinent patients dry the following methods may be found to be useful. In the case of a female a bidette is fitted, and in that of a male a length of colostomy tubing is fixed over the penis with strapping, the urine collecting in a receptacle placed at the side of the bed

The Skin, Mouth and Eyes —The skin needs early and regular care, otherwise it will soon be broken. For this reason, as well as for convenience of examination, it is important to strip a patient immediately and not to let him lie in his clothes until

prognosis becomes obvious. A difficult problem arises in a violently restless patient If abrasions are to be avoided, the knees, elbows and heels must often be bandaged in wool pads. The mouth should be kept clean to avoid parotitis, and this is best done by winding a wisp of wool around a stout pencil of wood and by swabbing the inside of the cheeks, the teeth and tongue with a mixture of glycerine and borax water. Metal forceps must not be used, because the patient may clench his jaws and break his teeth. Also, the finger should not be used, as this may be severely bitten.

When eyelids are swollen the conjunctival sacs should be irrigated regularly with boracic lotion

Temperature—It is usual to place a shocked patient in a radiant-heat cradle. With proper precautions this is a wise measure, but a warning must be given, since the thermoregulating centres are often temporarily in abeyance and the body tends to take on the temperature of its environment. An excessive amount of heat may therefore be applied, which not only causes an undesirable loss of fluid by sweating but also may lead to hyperthermia. These complications may be avoided if the patient's temperature is taken at regular intervals.

A rise in temperature of 2° F or more should be treated by tepid sponging, or by cold packs and intravenous injections of aspirin (20 gr) if the temperature cannot be controlled by other means.

Absorption of blood in subarachnoid hæmorrhages often causes a rise in temperature of 1° to 2° F.

Hypostatic pneumonia may occur, with or without obvious signs referable to the chest, when a rise in temperature may be the only sign of its onset A rigor must be reported by the nursing staff at once, as this often indicates the onset of meningitis. Immediate chemotherapy in such cases is necessary if the patient's life is to be saved. Severe intrinsic injuries, particularly of the hypothalamus, commonly lead to death in hyperthermia, for which no kind of treatment is of any avail.

The First Twelve Hours—Primary shock is treated by warmth and rest, and in the majority of cases no other treatment is needed. Apart from complications elsewhere in the body, loss of blood in the immediate stages of a closed head injury is usually so small that blood transfusions are rarely necessary In those cases when the blood pressure remains persistently low (100 mm. Hg. and below), a small amount of blood (250 c c.) may be given with advantage, and this may have to be reinforced by suitable stimulating drugs These, however, are usually better avoided.

Urgent associated injuries, such as rupture of abdominal viscera, must be treated irrespective of the degree of concussion, for the obvious reason that the patient will die if this is not done.

Fractures should be splinted in the first few hours to prevent further deformity, but on no account should they be forcibly manipulated as this seriously aggravates shock. Excision of compound fractures may safely be left for eighteen hours if the condition of the patient is such as to necessitate the delay, and particularly if the operation cannot be done under local anæsthesia. It cannot be too strongly stressed that general and even spinal anæsthesia is extremely dangerous in the early stages of concussion, since any further change in intracranial tension or blood pressure may render inadequate a cerebral circulation which is already embarrassed by pathological changes within the skull.

After Twelve Hours.—A lumbar puncture is performed and a measurement taken of the cerebrospinal fluid pressure. When the pressure is below 150 mm. nothing further need be done, as this lies within normal limits. When it is above 150 mm., fluid should be withdrawn until the pressure is reduced to 50 mm. This figure was chosen because it is the minimal normal and therefore allows of the maximal accumulation of cerebrospinal fluid before an abnormally high pressure is reached again. When the cerebrospinal fluid is heavily stained with blood it should be freely withdrawn at four-hourly intervals until active bleeding, as shown by cell counts, has ceased.

After Eighteen Hours.—Whether a patient is improving or not a lumbar puncture should be performed at the end of eighteen hours and sufficient fluid withdrawn to bring the pressure to 50 mm. Should a pressure above 200 mm. be found at this second reading, spinal drainage will have to be reinforced by some other kind of dehydration, and the choice of the method will depend on the condition of the patient's circulation. If the pulse is full and bounding, 3 oz. of magnesium sulphate dissolved in 6 oz. of water should be introduced slowly into the rectum by means of a funnel and tube and great care taken to see that it is retained. When the circulation is feeble or normal, 75 c.c. of 50 per cent. solution of sucrose should be injected intravenously. A profuse subarachnoid hæmorrhage, however, is a contraindication to intravenous therapy, since further bleeding may be aroused if the blood pressure is raised.

The general metabolic processes must also be considered as well as the local conditions inside the cranium. Dehydration, therefore, must not be carried to the extent of damaging the other tissues of the body in an attempt to overcome hypothetical cerebral œdema or excessive secretion of cerebrospinal fluid. Hypertonic salines are also contraindicated in old people whose tissues are already dehydrated.

After Twenty-four Hours.—The cerebrospinal fluid pressure

is checked every twelve hours until the patient is conscious, and kept within normal limits by lumbar drainage, reinforced if necessary by dehydration. Fluids are given in sufficient quantities to maintain the circulation, and blood transfusion also will be necessary should the patient's pulse begin to deteriorate.

Comment.—Treatment of closed injuries of the brain in the acute phases is arousing much controversy. Most American surgeons interested in cerebral trauma advise early and vigorous intravenous dehydration, and much has been written on this subject by Temple Fay.[1] British surgeons, on the other hand, usually oppose dehydration in the early stages, Jefferson[2] stating that it should rarely be used before the second day. There is no doubt that it is not without danger. Obviously it should not be employed until the intracranial pressure has been proved to be supranormal by spinal manometry, and, if possible, the cause of the rise of pressure determined.

The objections to intravenous dehydration are as follows :—

1. It may increase bleeding.
2. It may interfere seriously with the general metabolic processes of the body.
3. The tissues of the body may be over-dehydrated with resulting toxæmia.
4. Lumbar puncture may be impossible in a restless patient, and in these cases it is wrong to dehydrate on the assumption that the pressure is raised.
5. There is no proof that cerebral œdema is an important factor in the production of symptoms in the early stages of trauma.
6. Theoretically it is inconsistent to dehydrate and to give fluids by mouth.

My practice lies between these extremes, for although I believe that control of intracranial tension is essential if the best results are to be obtained, the general requirements of the body must also be carefully watched and respected. There can be little objection to withdrawals of cerebrospinal fluid by lumbar puncture controlled by manometry, and this, in my experience, is the best method of keeping intracranial pressure within normal limits.

SURGICAL TECHNIQUE

Subtemporal Explorations and Decompressions.—The term subtemporal decompression, by common usage, has come to

[1] Fay, T "The Treatment of Acute and Chronic Cases of Cerebral Trauma by Methods of Dehydration" *Ann Surg*, 1935, **101**, 76-132

[2] Jefferson, G "Remarks on the Treatment of Acute Head Injuries" *Brit Med Jour*, 1933, 2, 807

mean the removal of bone beneath the temporal muscle but does not necessarily imply that an opening is also made in the dura mater. To avoid ambiguity, the term " decompression " is here used to denote that the dura mater has been widely opened to relieve tension within the dural envelope · and " exploration," that the bone only has been removed for purposes of inspection of the extradural space A decompression must always be made over a silent area, since the part of the brain uncovered is apt to herniate through the dural opening and to become strangulated or contused with consequent loss of function. A left-side subtemporal decompression is not entirely without danger, as an aphasia might ensue. Therefore whenever the clinical indications do not demand an exposure elsewhere, a decompression should always be made on the right side beneath the temporal muscle and over the temporal lobe of the brain.

SPECIAL INSTRUMENTS AND MATERIALS (Fig 53)

1 Safety-razor
Lintine and dental swabs
1 Toothed and 1 non-toothed dural dissecting forceps
1 Curved dissector (Adson type)
1 Rougine
1 Teaspoon
1 Saline syringe (C Ryle)
2 Flat metal brain retractors
2 Self-retaining retractors
1 Michel clip apparatus
1 Hudson's brace (large handle pattern)
1 Perforator
1 Set of graded burrs
1 Trephine (author's pattern to fit brace)
1 Wilms forceps
1 Double-acting nibbling forceps
1 de Vilbiss forceps
2 Gigli saw handles
2 Gigli guides and saws
1 Sucker apparatus
1 Diathermy apparatus, leads and electrodes
1 Electromagnet
1 Overhead table or tray
1 Silver clip apparatus
6 Rubber bands
Horsley's wax
Waxed silk
12 No 15 Bonney's needles (half-circle)
1 Headlight
At least 3 dozen fine-pointed hæmostats

Preparation of the Scalp.—The hair is cut short with scissors or clippers and the whole head is shaved with a safety-razor, great care being taken not to break the skin A skilled barber, of course, may use any method he wishes. After shaving, the scalp is washed and scrubbed with soap and water and then swabbed with perchloride lotion. Finally, it is treated with spirit and wrapped in a sterile compress. Iodine should not be used as it is apt to scale or burn the skin. Careful preparation of the scalp is important if infection is to be eliminated, and complete shaving is also necessary, since it is impossible to know before operation whether both sides of the skull will have to be opened or not

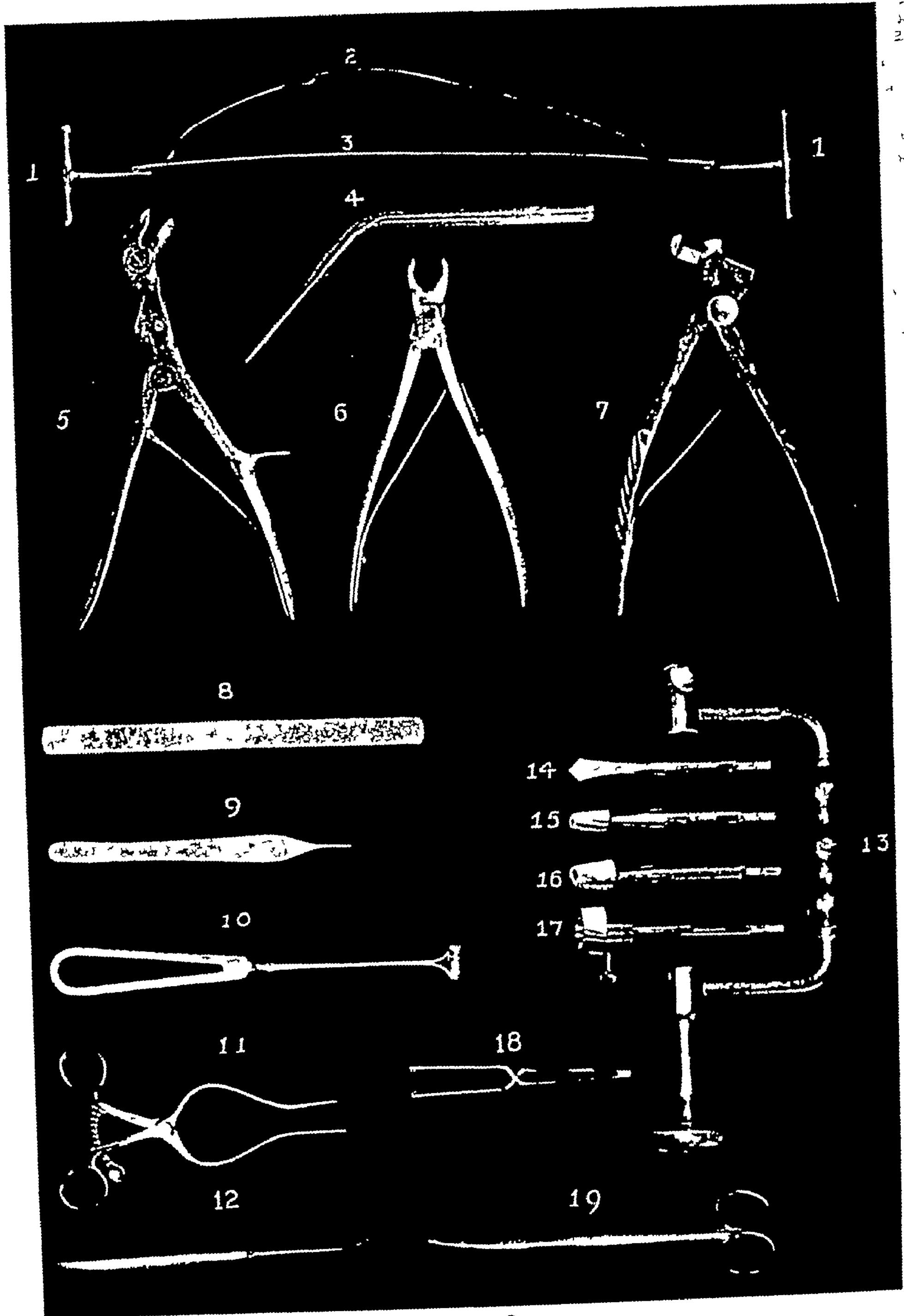


Fig 53

Special instruments

1, Gigli saw handle
2, Gigli guide
3, Gigli saw
4, Sucker end
5, Double-acting nibblers
6, Wilms forceps
7, de Vilbiss forceps
8, Brain retractor
9, Dural dissecting forceps
10, Rake retractor
11, Self-retaining retractor
12, Adson's curved dissector.
13, Hudson's brace
14, Perforator
15, Burr
16, Burr
17, Trephine to fit brace
18, Michel clip applicator
19, Dural scissors

Anæsthesia.—Operations for closed head injuries should be done whenever possible under local anæsthesia When, for reasons of restlessness, a basal or general anæsthetic or a combination of the two has to be given, the chances of recovery are seriously diminished.

When a patient is sufficiently unconscious to tolerate intubation without general anæsthesia, an intratracheal tube lubricated with percaine ointment (10 per cent.) should be introduced and connected through a suitable apparatus with an oxygen cylinder. This precaution will often save the lives of those patients whose respiratory centres fail before there has been time to relieve the compression of the brain. Care in these cases must be taken not to overdistend the lungs by blowing in oxygen too rapidly.

Restlessness under local anæsthesia may be due to (1) suffocation, (2) overheating under wet towels, (3) pain in the wound owing to inadequate anæsthesia and (4) postural discomfort. Before resorting to drugs to quieten a patient each of these factors must be reviewed and corrected if necessary. When a patient continues to be restless for reasons other than those of faults in operative technique, an intravenous injection of pentothal, evipan or morphia is necessary. In my opinion, pentothal in the hands of an expert anæsthetist is an excellent anæsthetic.

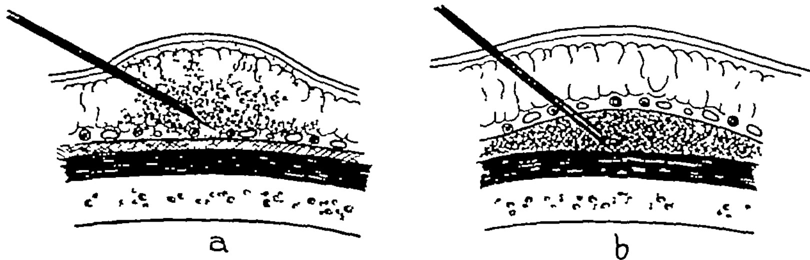


Fig 54

a, Correct **novocain infiltration of scalp** The solution is diffused in fibro-fatty tissue and acts on larger nerves and vessels in its deepest layer *b*, Incorrect infiltration The needle has passed too deep and its point lies in subaponeurotic areolar tissue, where the solution is ineffective on scalp as aponeurosis is relatively impervious (*Norman Dott*)

The line of an incision is infiltrated with novocaine-adrenalin solution until the skin is raised into a distinct mound. Whether the anæsthetic is injected above or below the galea appears to make very little difference to the resulting anæsthesia, but as Dott has pointed out, the correct anatomical layer is superficial to the galea, as it is there that the nerves are found (Fig. 54). The needle is then inserted at right angles to the bone and the pericranium injected at intervals of 1 in. Further injections are made according to the nerve supply of the part to be exposed, and these details will be given later when the various operations are described. A generous amount of anæsthetic should always be used, up to

100 c.c. of 1 per cent. novocaine solution to which 5 drops of adrenalin (strength 1 in 1,000) have been added may be injected with safety. At least five minutes should be given to allow the novocaine to act before making the incision, because a painful start may rouse the patient and make him restless for the remainder of the operation. Further injections of anæsthetic are often necessary in the later stages of an operation, particularly when the wound is being closed. Local anæsthesia should not be omitted in a deeply unconscious patient because an unblocked incision will inflict further shock. Moreover, a patient is apt to become more conscious as a compression is relieved, and resentment of a painful unanæsthetised wound may be shown by uncontrollable restlessness.

When general anæsthesia is unavoidable on account of restlessness, gas and oxygen reinforced by chloroform administered through an intratracheal tube is a useful method. Ayre advises that intratracheal chloroform only should be used if advantage is taken of the special tube designed by himself.[1] At no stage in the operative proceedings must the patient be allowed to become cyanosed. Basal anæsthetics such as avertin, I believe, are dangerous and are better avoided. The administration of ether and chloroform by the open method cannot be too strongly condemned, because such anæsthesia increases intracranial pressure and venous congestion to a degree that makes intracranial manipulations impossible or exceedingly dangerous. Moreover, it is undesirable to have an explosive vapour like ether in the air when a diathermy current is being used.

General Theatre Technique.—Correct posturing and towelling on the operating table are so important that they can make all the difference between a successful and unsuccessful operation.

The patient is placed supine or prone on the table according to the part of the skull which it is proposed to open. For example, in a right-sided subtemporal decompression he should lie on his back with the right shoulder raised and his face turned to the left side. He is strapped into position to prevent him from slipping off the table should be become unruly, and his wrists are loosely tethered to avoid the danger of his putting his hands into the wound. An easy way to secure the patient is to pass folded sheets over the chest and thighs, and after overlapping their ends under the table, to fix them in position with a line of safety-pins. The hands can be controlled by a bandage turned into a clove-hitch knot passed over the wrists and then attached to the sides of the table. The limbs should rest in a comfortable position and painful pressure points should be avoided by padding the table with pillows. The head is raised above the rest of the body to promote

[1] Ayre, T P "Anæsthesia for Cranial Surgery" *Lancet*, 6th March 1937

venous drainage and the operation field should be easily accessible to both the surgeon and his assistant (Fig 55). A linen towel over a square of batiste is placed beneath the head, a large lint guard wrung out in perchloride lotion is folded over the scalp and four large towels wet with perchloride lotion are used to shut off the operation field The ends of the towels towards the face are lifted from the forehead and draped across an overhead table or tray so as to keep the patient's nose and mouth free This ensures a free airway. Finally, a large sheet with an opening in

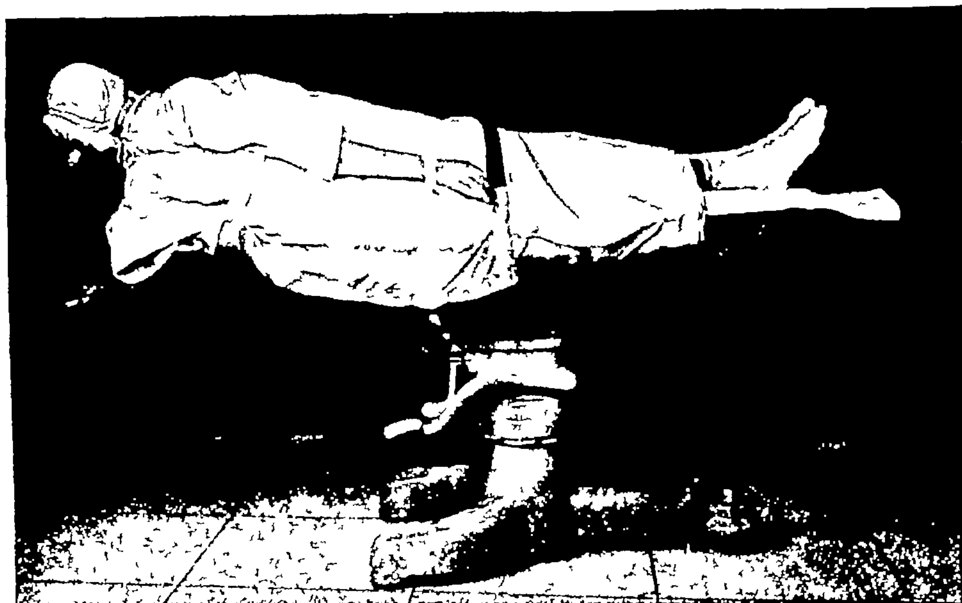


Fig 55
A patient placed in position on the operating table for a subtemporal decompression under local anæsthesia

one end is used to drape the whole field The surgeon and his assistant stand on each side of the patient's head and the two theatre nurses are placed on raised stools at each end of the overhead table (Figs. 56-59) If the towels are clipped to the lint guard and the guard sewn to the anæsthetised edge of the wound, the towels will follow any movement of the patient's head should he become restless.

The Operation of Subtemporal Exploration by a Muscle Split.—The skin incision is marked out with an iodine line It starts at the upper border of the zygoma 1½ in. in front of the external auditory meatus and runs upwards for 2½ in, then curves horizontally backwards for ¾ in The classical incision, consisting of the vertical limb only, gives an inadequate exposure, particularly as the temporal muscle is often thickened with blood and consequently so rigid that mobilisation is difficult

Local anæsthetic is injected along the line of the incision and

FIG 56 —An overhead operating table (Author's design)



FIG 57

Position of the patient under the overhead table Note how the towels are draped away from the face, ensuring a free airway Note also the position of the anæsthetist

Fig 58

A method of towelling



Fig 59

An operating team in position

along its base from the margin of the orbit anteriorly to the mastoid bone posteriorly, particular care being taken thoroughly to anæsthetise the tissues above the ear. The needle is then inserted at various points at right angles to the skin and the

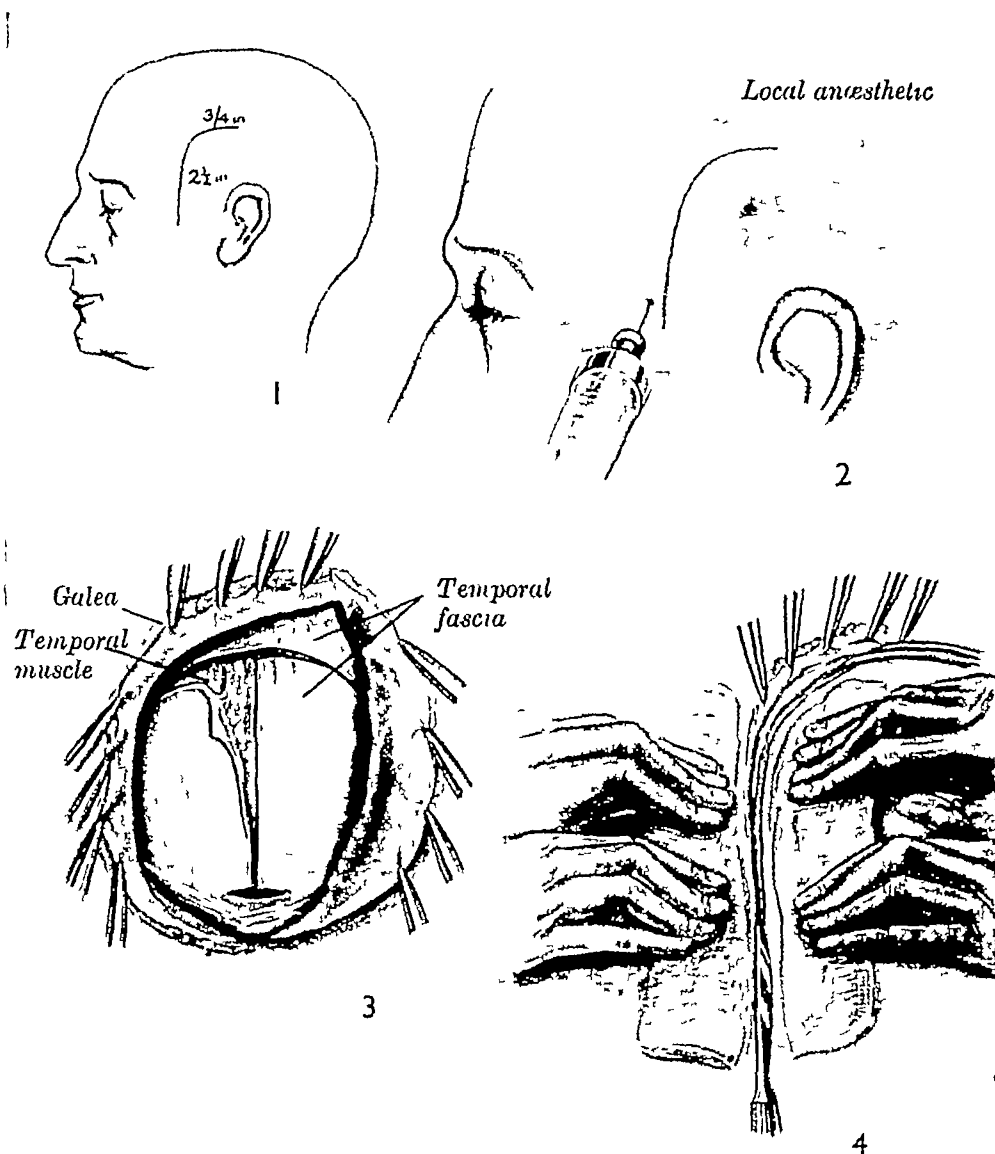


FIG 60
Subtemporal exploration by muscle split
1, Line of incision 2, Zone of anæsthesia
3, Method of reflecting the temporal fascia
4, Exsanguination of the skin by digital compression as an incision is made

lower part of the temporal muscle is freely infiltrated down to the bone so as to anæsthetise the deep temporal nerves as they turn upwards. If a generous amount of anæsthetic is used the whole of the temporal fossa can be made completely insensitive.

Each side of the incision is then compressed by the finger-tips

of the assistants to control bleeding and to pull the wound apart as the skin and deep fascia are incised. The galea is picked up with artery forceps at intervals of ½ in. It is unnecessary to secure each bleeding point separately, since the drag of the artery forceps

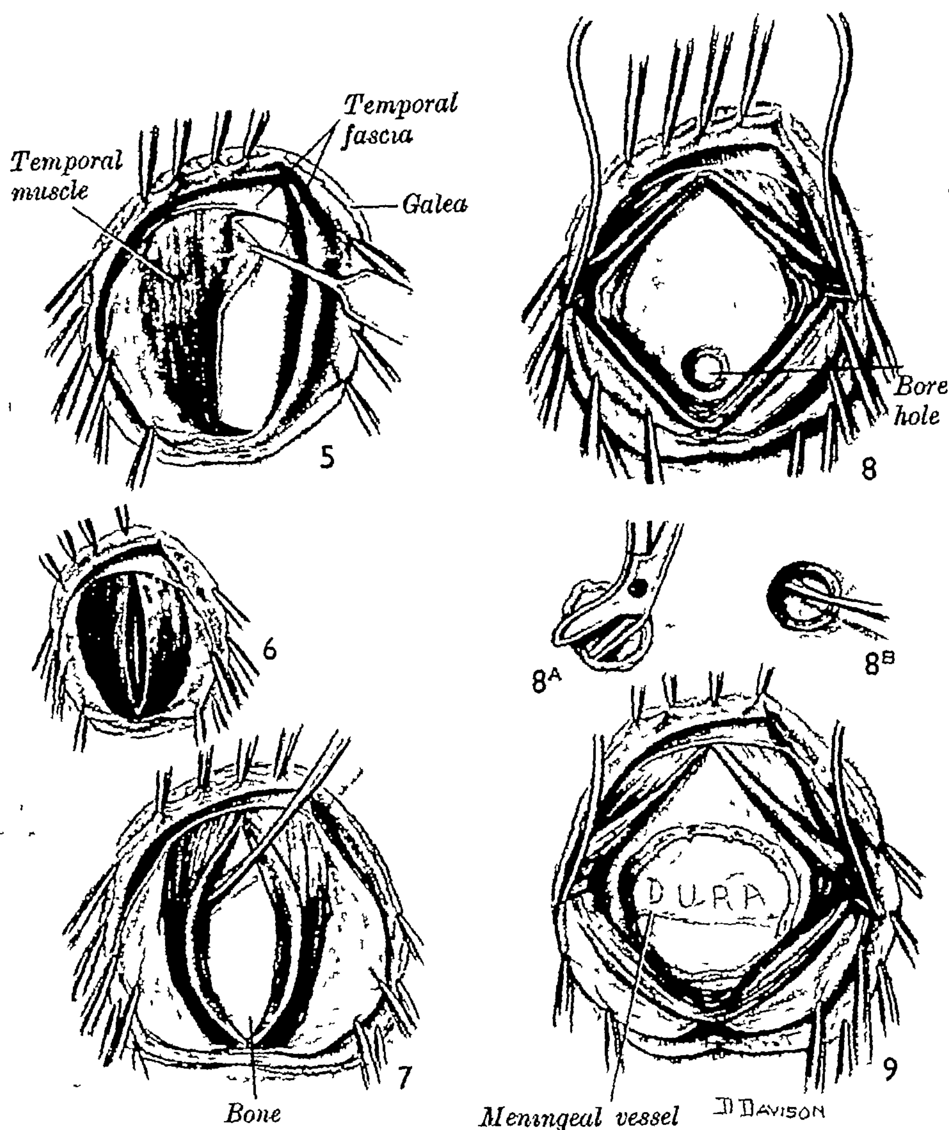


Fig 61

5, Exposure of the temporal muscle
6, Splitting the temporal muscle
7, Separation of muscle from bone
8, Exposure of bone
8A, Removal of bone by nibbling
8B, Separation of dura from bone
9, Exposure of dura and middle meningeal vessels

will produce satisfactory hæmostasis when they are thrown over the edges of the wound The triangular skin flap is reflected backwards and the skin at the anterior end of the wound is undermined. The artery forceps are arranged into neat bundles by elastic bands and pinned to the towels so that they do not obstruct the operation field. An alternative method of hæmostasis is to

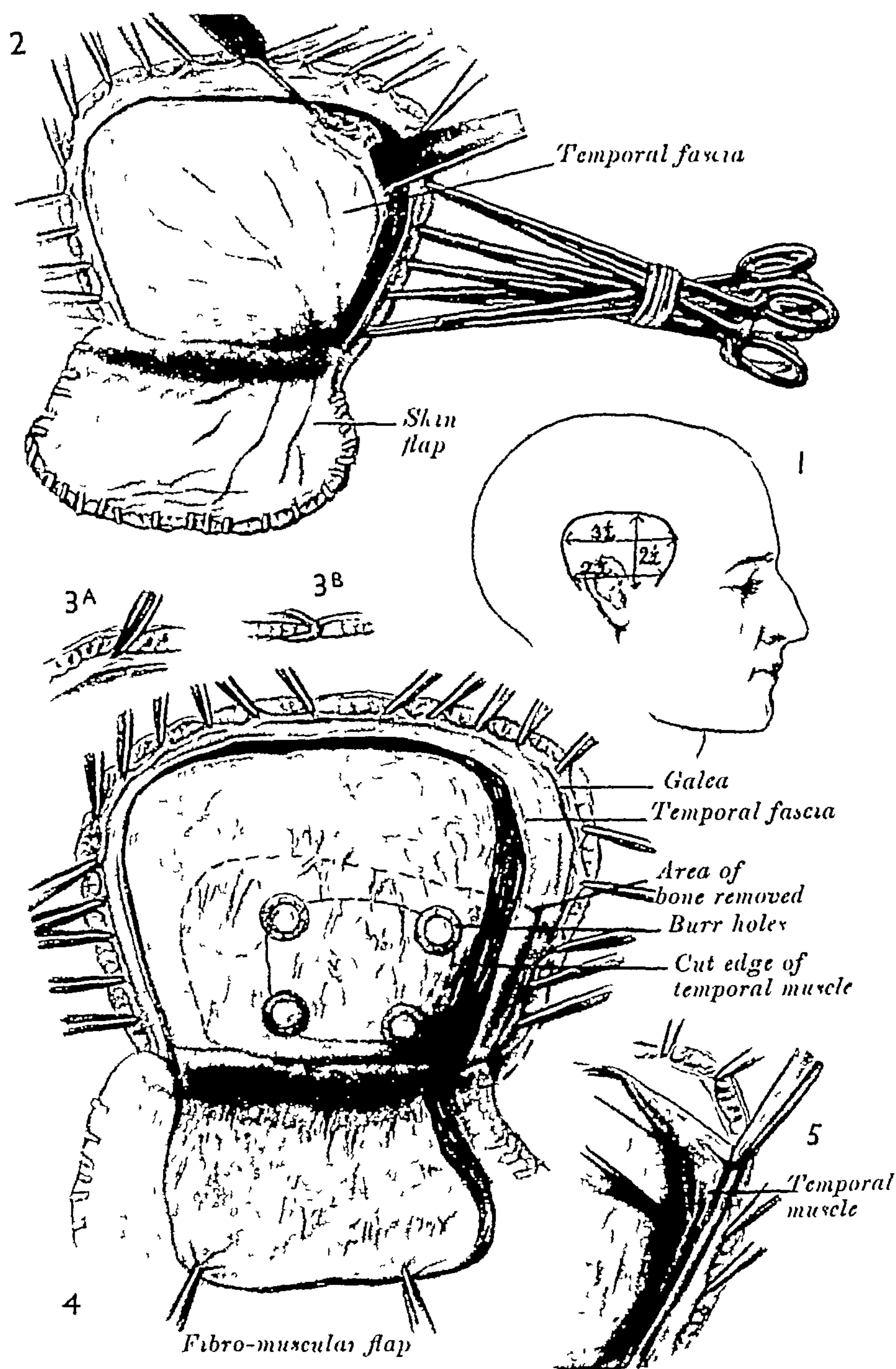


FIG 62

Subtemporal decompression by a muscle slide

1, Line of skin incision and zone of local anæsthesia
2, Subperiosteal separation of the temporal muscle from the bone after the skin flap has been reflected
3A, Method of securing hæmostasis in the skin by hæmostatic forceps clamped to the galea
3B, Method of securing hæmostasis in the skin by Michel clip
4, Proposed area of bone removal after reflection of the skin and temporal muscle
5, Undermining of the anterior and attached part of the temporal muscle to permit of removal of the bone overlying the tip of the temporal lobe of the brain

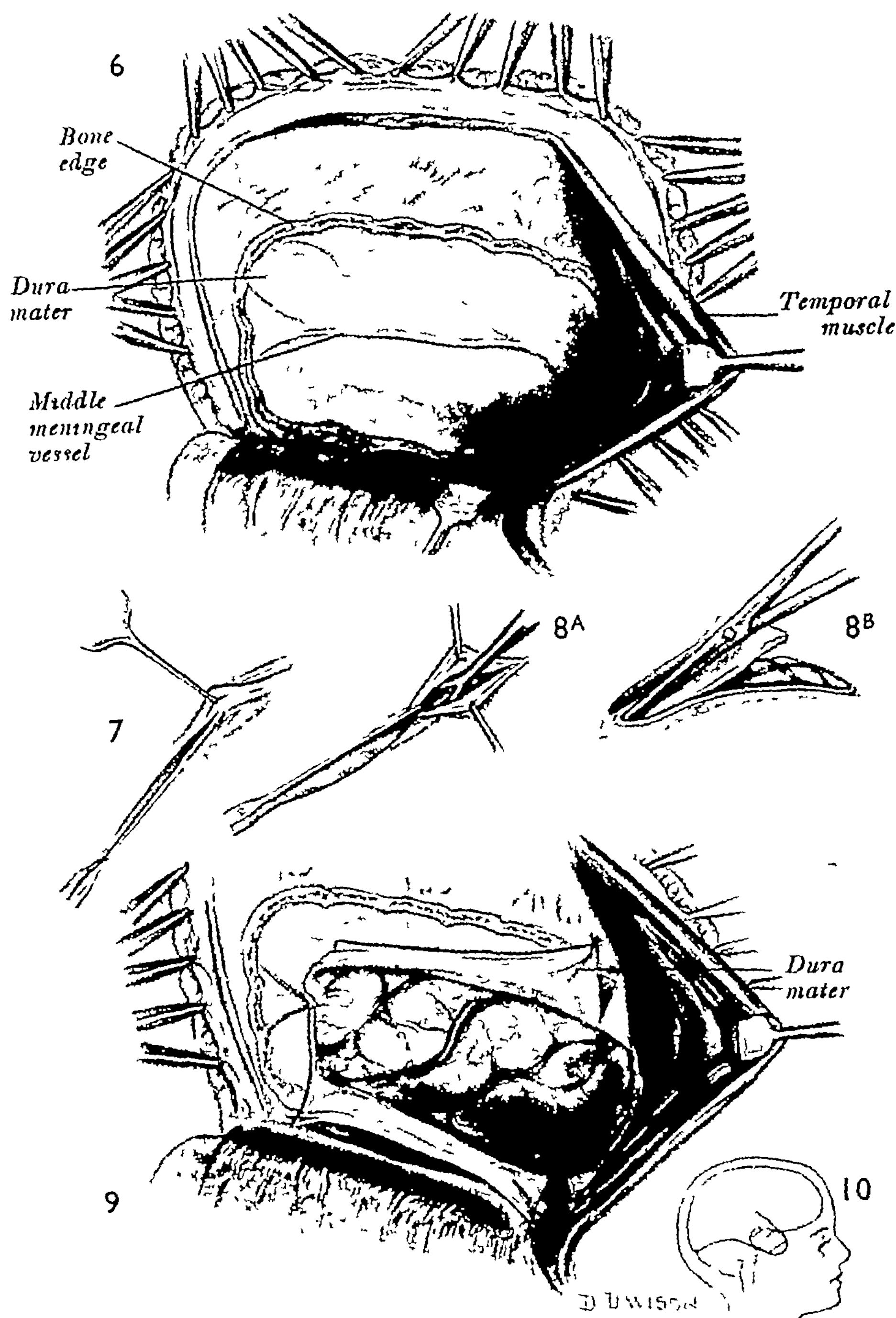


Fig 63

Subtemporal decompression by a muscle slide

6, Completion of the bone removal 7, Preliminary incision of the dura mater

8A, Method of enlarging the dural incision by means of a scalpel with a curved dissector to protect the cortex

8B, Method of enlarging the dural incision by means of scissors with a strip of wet lintine to protect the cortex

9, Brain decompressed with dura mater sutured to the pericranium to prevent formation of post-operative clots due to oozing from meningeal vessels

10, Inset shows area of bone which must be removed if the decompression is to act efficiently

clamp tetra cloths into position with a series of closely placed Michel clips.

By means of an incision shaped as shown in Fig. 60 the temporal

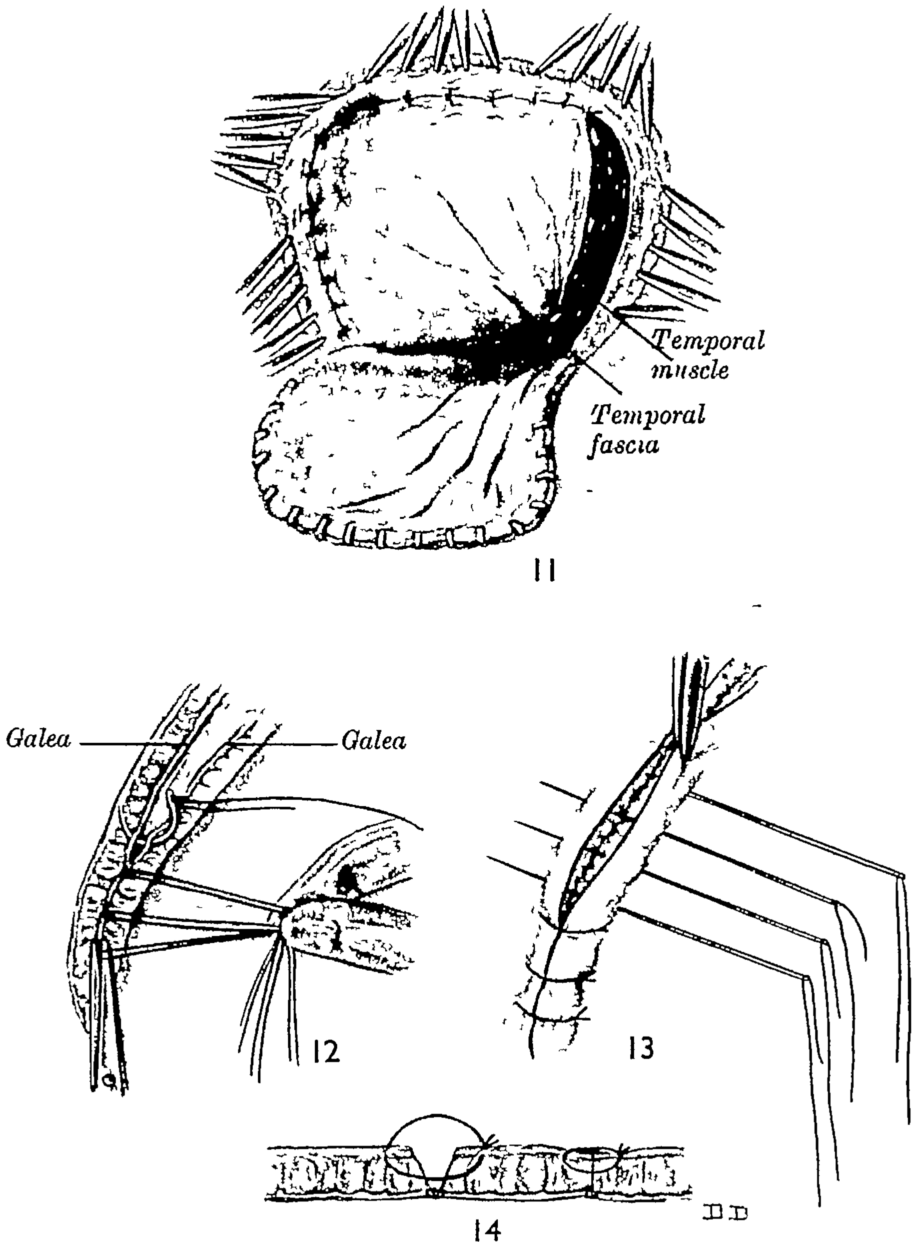


FIG 64

Subtemporal decompression by a muscle slide

11, Method of suturing the fascia and muscle The fascia is not sutured anteriorly
12, Introduction of the galeal sutures
13, Repair of the skin
14, Skin and galeal sutures in position

fascia is separated from the temporal muscle by sharp dissection and turned backwards and forwards as two small flaps The small horizontal incision above the zygomatic arch, shown in

Fig. 60, is important if adequate access is to be obtained to the base of the skull. The temporal muscle is split for the whole length of the exposure in the line of its fibres and then is freely separated from the bone with a curved dissector. One or two self-retaining retractors are now inserted to hold the muscle apart and an area of bone about 1½ in. in diameter should be exposed. A burr hole is made, and after the dura mater has been pushed away with a curved dissector the bone is removed by nibbling forceps in the whole of its exposure down to the base of the skull (Fig. 61). The technique of opening the bone is described below.

The Operation of Subtemporal Exploration by Muscle Slide (Figs. 62-65).—The incision is shaped like a horseshoe. It starts at the middle point of the upper margin of the zygomatic arch and runs upwards and forwards for 2½ in., then it curves backwards for 3½ in. and finally runs downwards and forwards to end behind the ear on the base-line. Novocaine is injected along the line of the incision and along the base of the flap from the external angular process of the frontal bone to a point 1 in. beyond the posterior end of the margin of the mastoid bone. The whole of the lower part of the temporal muscle is infiltrated down to the bone.

Digital compression on both sides of the wound is made by the assistant to control bleeding while the incision is made in sections, first through the skin and deep fascia. Artery forceps or Michel clips are applied as in the previous operation. The temporal fascia is now incised ¼ in. within and parallel to the skin incision, and the fibres of the temporal muscle in the anterior vertical limb are split down to the bone. The fibro-muscular flap is separated from the bone with a sharp rongeur and turned downwards with the skin flap. Sufficient bone is then removed to give adequate access to a bleeding meningeal vessel or to allow for a decompression.

The Method of opening the Bone (Fig. 65).—The disc at the top of the Hudson's brace is placed in the palm of the left hand and the handle is taken between fingers of the right hand. With the perforator screwed in position a funnel-shaped hole is made into the bone until the pale blue of the dura mater or the dark blue of an extradural clot is just seen; to continue further is dangerous, as the dura mater may be pierced and the cortex lacerated. The perforator is replaced by burrs of increasing size until a cylindrical hole has been made. When using the burrs a certain amount of thrust from the left arm is necessary, but the shoulder muscles must be so locked that should the bone give way suddenly the brace is under control and is not allowed to plunge into the brain.

It is important to stop turning the brace when the bite on the burr increases suddenly, as this means that the inner table has been pierced. A thin flake of bone is usually left at the bottom of a burr hole, but this is easily pulled or scraped away. The next

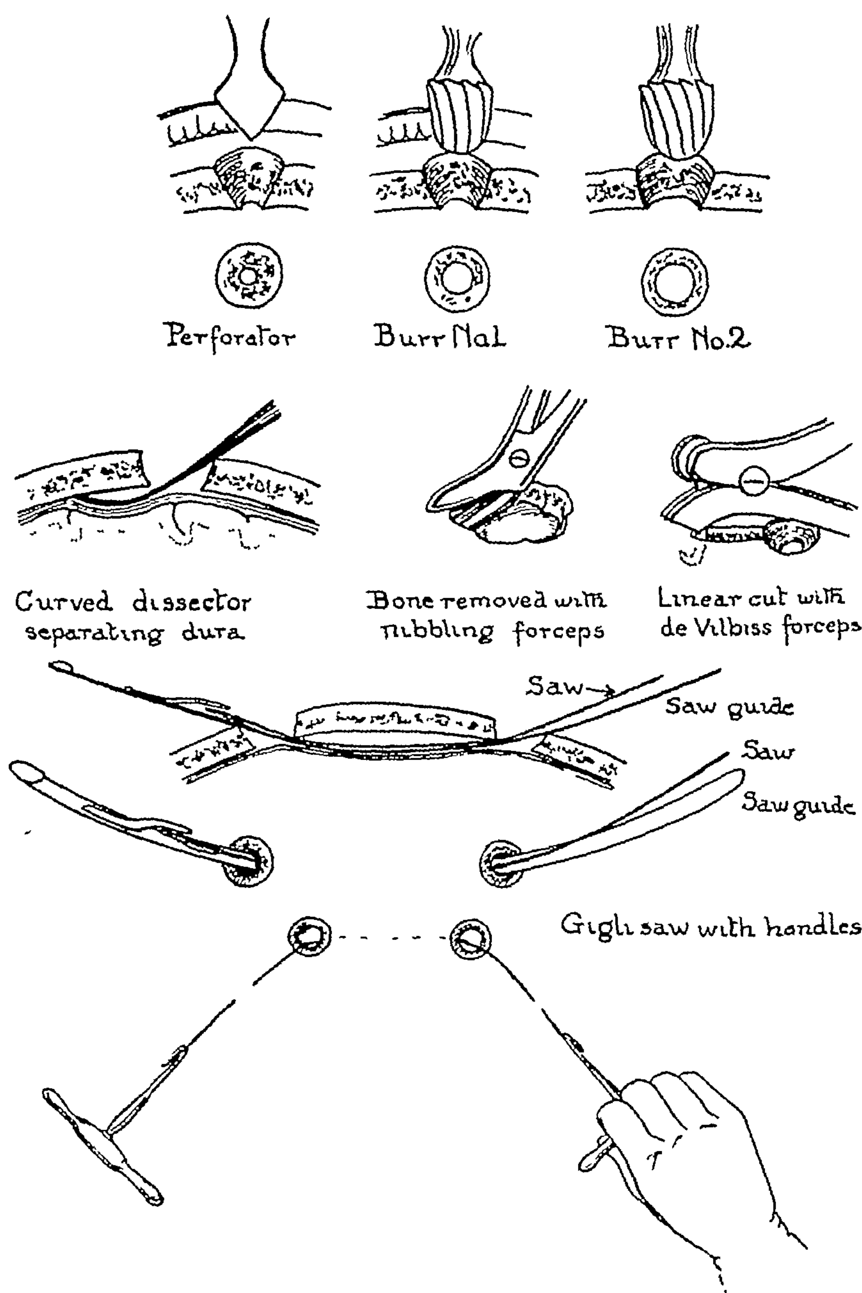


FIG 65
How to open the bone

step is to push the dura mater gently from the bone with a curved dissector so that it is not torn when the nibbling forceps are inserted. It is much safer to bite the bone away cleanly with forceps than to break it by twisting and leverage, because of the

danger of fracturing the bone beyond the limits of the proposed removal and because the forceps may break or slip and contuse the brain. In the limited exposure of the muscle-split operation one burr hole only is necessary, followed by nibbling In the wider exposure of the muscle-slide operation four holes are made and linked up by piecemeal nibbling. An alternative method is to join up the holes by linear cuts either with a de Vilbiss forceps or with a Gigli saw The bone is removed in the lower half of the exposure, including the part under the attached portion of the temporal muscle. It is essential that bone should be removed to the base of the skull if a decompression is contemplated.

How to deal with Extradural Hæmorrhage.—Extradural hæmorrhages are usually found in the form of large clots, and very little further bleeding takes place as long as they are left undisturbed. When the bone has been removed it is wise to wait for a few minutes to allow the cerebral circulation to readjust itself to the new conditions. It is extremely dangerous to insert a finger into the wound in an endeavour to hook out the clot, as troublesome bleeding may be induced before one is in a position to control it Moreover, the extra pressure of the finger superimposed on the already existing compression of the clot may prove fatal. The clot should be removed slowly a little at a time from above downwards with a suitable instrument, such as a teaspoon or a curved dissector, until the ruptured meningeal vessels come into view. The bleeding vessels are most easily sealed with the coagulating diathermy current, but if this is not available they may be underrun with a fine suture and ligatured, or compressed with a silver clip. The vessels rupture usually at a point above the base of the skull and are easily accessible Rupture on the base of the skull is much less common, and the necessity of plugging the foramen spinosum with a wedge of matchstick or with bone wax to stop bleeding is fortunately rare To do this successfully an efficient headlight is essential and the temporal lobe must be raised with flat brain retractors

Profuse bleeding which seems to come from many angles is usually not due to rupture of the meningeal vessels, and its control often presents most difficult technical problems. It may come from diploic veins in a fractured bone or from a ruptured venous sinus Horsley's wax should be pressed firmly into the crack of any fracture that can be felt or seen

When, in spite of careful waxing, bleeding continues and streams from above, it is probably coming from the superior longitudinal sinus.

In these cases, even though direct vision of the sinus through the subtemporal exploration is impossible, it is unwise to make a

further opening near the midline to try and expose the tear, since its exact location is not known. It is usually safer to cut small grafts from the temporal muscle and to pack these towards the bleeding point between the dura mater and the bone with a pair of long dissecting forceps. When this has been done, small stitches should then be passed through the dura mater at the upper edge of the exposure and taken over the bone and through the pericranium, so that when the sutures are tied the drag on the dural envelope will keep the grafts in position. When the bleeding comes from the base, muscle grafts should be packed towards the petrosal, sphenoidal and cavernous sinuses. Occasionally it may be necessary to use strips of gauze instead of muscle grafts. When this is done they should be removed forty-eight hours later under direct vision with the wound widely open. If they are withdrawn blindly through a small opening, serious bleeding may start again.

Opening of the Dura Mater for Decompression of the Brain.—When the brain is under tension, opening of the dura mater can be a very difficult manœuvre, and the greatest care must always be taken not to bruise the brain or wound the cortical vessels. The opening is made as high as the Sylvian fissure but no higher, otherwise the lower end of the motor cortex will be endangered On the other hand, it must go down to the base of the skull if the decompression is to act efficiently. A cross-incision will be found most useful in the muscle-split operation, but in the wider exposure produced by a muscle slide a rectangular or semicircular flap may be turned upwards. With a sharp scalpel an incision ½ in. long is gradually deepened until a curved dissector can be inserted between the arachnoid and dura. The incision is then enlarged by cutting down on to the blade of the dissector An alternative method of continuing the incision is to cut the dura with scissors after direct vision has been obtained by depressing the brain for a few millimetres with a strip of wet lintine pushed into position with a pair of narrow-bladed dissecting forceps (see Fig 63).

Methods of controlling Bleeding (Figs. 66 and 67)—As the circulation of the brain is already embarrassed, loss of blood during an operation is dangerous Care, therefore, must be taken to have any possible bleeding under control before any manipulation in an operation is begun, and in particular, digital compression of the skin must never be omitted as the first incision is made

A coagulating diathermy current is used for sealing veins or small arteries in the muscle, dura and brain The neatest and most convenient method is to pick up the vessel concerned between the blades of a finely pointed dissector, and as the field is dried

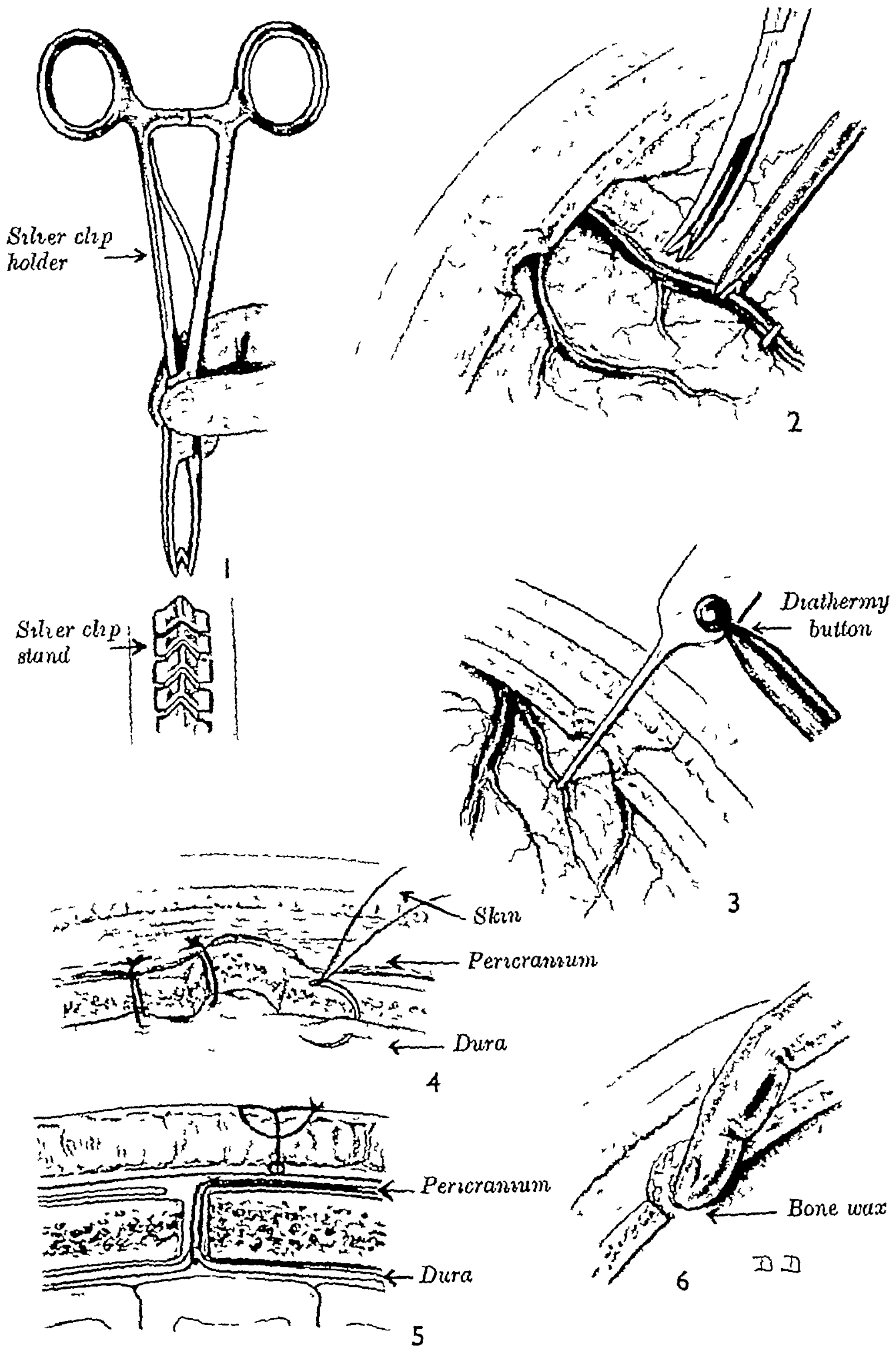


Fig 66
Methods of controlling bleeding
1 and 2, Compression by silver clips 3, Diathermy coagulation
4 and 5, "Hitching" of dura to pericranium to prevent extradural clots
6, Plugging of diploic vessels with Horsley's wax

with the sucker, to touch the dissector with the diathermy electrode. A warning, however, is necessary, for although the diathermy current is so useful in controlling bleeding in the deeper tissues it must never be used on or superficial to the galea, otherwise the skin edges will necrose and the wound will not heal by first intention. Care must also be taken that the artery forceps are not touched inadvertently, otherwise the same complication will occur.

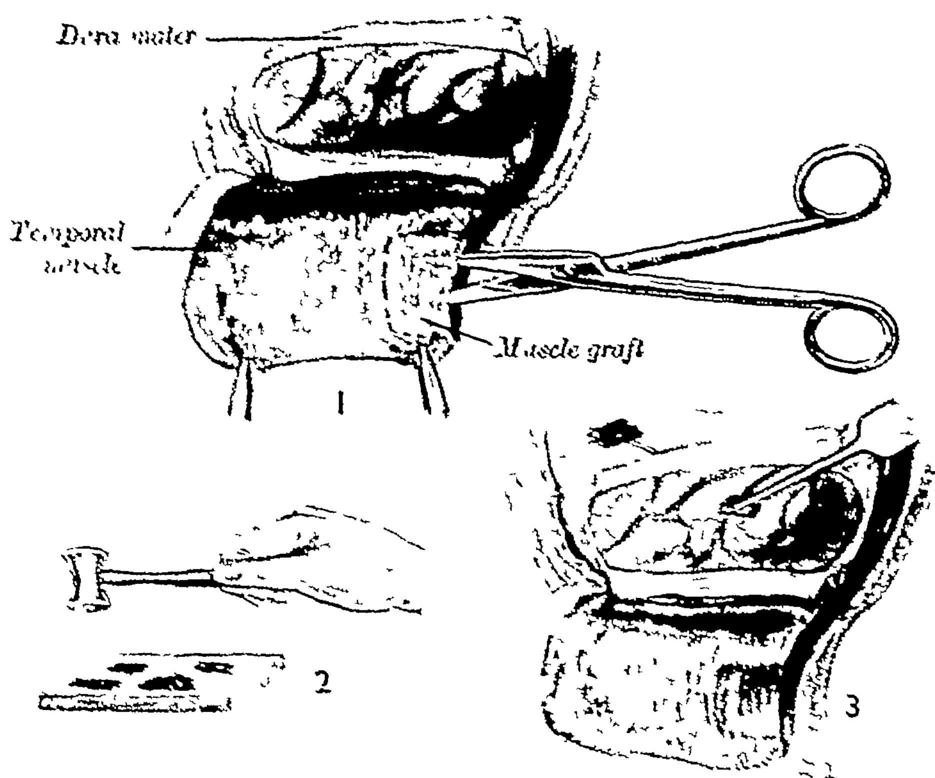


FIG. 67

Preparation and application of muscle grafts.

1. Cutting graft from temporal muscle. The whole thickness of muscle is not used
2. Hammering of muscle grafts into thin sheets.
3. Grafts in position on the cortex and dura.

When a large artery is bleeding or has to be divided, it is safer to compress it with a small silver clip applied on a special holder. Ligature of the delicate cerebral vessels is always a lengthy process and often difficult.

Hæmorrhage from bone is easily controlled by pressing Horsley's wax firmly into the bleeding channel.

Muscle grafts are of particular value in the repair of tears in the venous dural sinuses. They are also useful in controlling persistent bleeding from the surface of the brain when other

methods have failed. A flat piece of muscle cut from the temporalis and hammered into a fine sheet will stick like a postage stamp when placed in position.

Before any wound is closed it must be left completely dry as far as bleeding is concerned, otherwise a post-operative clot will form. It may be taken as an axiom that a bleeding cerebral vessel, however small, will continue to bleed and compress the brain before the tension in the clot rises sufficiently to occlude it. Many post-operative hæmorrhages can be prevented by tacking the dura mater at the periphery of the wound to the pericranium with occasional interrupted sutures. Thin layers of clot on the dura itself are best left in position, since they are unlikely to give rise to compression when the wound is closed. Also, they act as seals to numerous small vessels which otherwise would bleed

Wound Closure—*Muscle Split.*—The edges of the muscle are apposed in their whole thickness by interrupted sutures of fine silk or thread introduced on small curved needles.

The method of suturing the temporal fascia depends on whether or not the dura mater has been left opened. In decompressions the muscle only is drawn together so that its fibres can, if necessary, stretch apart and accommodate a bulge of the brain. When the dura mater has not been opened the fascia may be sutured, but as it often contracts during the operation it is usually impossible to repair more than its lower half.

Muscle Slide.—As the temporal fascia will slide to some extent across the muscle face, any natural retraction of the membrane during the stages of the operation can be easily overcome by the following manœuvre and an excellent covering for the bony opening obtained. The fascia is sutured above and behind but left open in front, here, only the muscle fibres are drawn together (see Fig. 64).

Buried sutures introduced at intervals of $\frac{1}{4}$ in. are used to appose the galea These sutures must never be omitted or replaced by deep through-and-through sutures, because the integrity of the wound depends on this layer. Care must be taken to cut the buried sutures accurately on the knots with fine scissors, otherwise long ends of silk will get between the skin edges and prevent healing. Correctly inserted galeal and skin sutures will control superficial vessels, and it is unnecessary to ligate each individually (see Fig 64).

When a wound is not under great tension the stitches may be removed in three days.

A sterile gauze dressing damped with spirit is placed over the wound and fixed in position by winding a sterile 6-in. gauze roll around and over the head. In a restless patient, when there is a

danger of his removing the dressing or of putting his fingers on to the wound, a stout linen bandage is also used. This is fixed under the chin but never wound round the neck, because of the danger of compressing the internal jugular veins and thus causing cerebral venous congestion with renewed bleeding (Fig. 68).

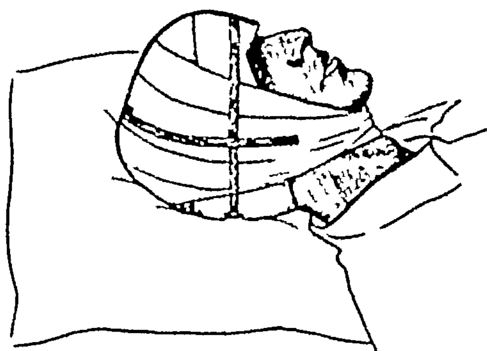


FIG. 68

Method of securing a dressing on a head wound. (*Norman Dott*)

Subdural Hæmorrhages—(*a*) *Acute.*—An acute subdural hæmorrhage may be diagnosed before operation, though it is usually found when making a subtemporal decompression in search of a middle meningeal hæmorrhage. Treatment is simple; the dura mater is opened to the full extent of the operative exposure and the clot is removed by suction; ruptured vessels are sealed and the subdural space is drained until all active bleeding ceases. If the ruptured vessel is not immediately apparent in the wound it is useless to search for it since the bleeding often comes from a distance. Moreover, it is dangerous forcibly to depress the cortex of the brain in an attempt to gain a wider exposure, because tethering veins may be torn. Occasionally subdural clots accumulate beneath the temporal lobes and can be demonstrated by encephalography only.

(*b*) *Chronic*—When localising neurological signs are present a 1-in. trephine hole is made over the presumed centre of the clot The advantage of a trephine hole over a burr hole is that the disc of bone removed may be replaced and the skull thus repaired if drainage at a particular site is not found necessary. This procedure, as will be shown later, will eliminate many difficult medico-legal arguments which otherwise might arise. When localising signs are absent, or when exploration is negative in the first instance, holes are sunk at various sites until the clot is found. Before abandoning the search for a clot, both sides of the skull must be explored at positions over the parietal eminences and just above the Sylvian points.

Clots are easily recognised by their black, dark blue or greenish-yellow colour. When they are present a cross-incision is made through the dura mater and the underlying adventitious membrane The fluid contents are evacuated by suction through a suitably curved nozzle. All accessible adventitious membrane should be removed by dissection When the resulting cavity is large, a second trephine hole should be made at a distant periphery, and

remaining fluid syringed out by injecting saline into one hole and letting it escape through the other. Finally, small drainage tubes are inserted and kept in position for forty-eight hours or longer according to the continuation of the discharge (Fig. 69). Occasion-

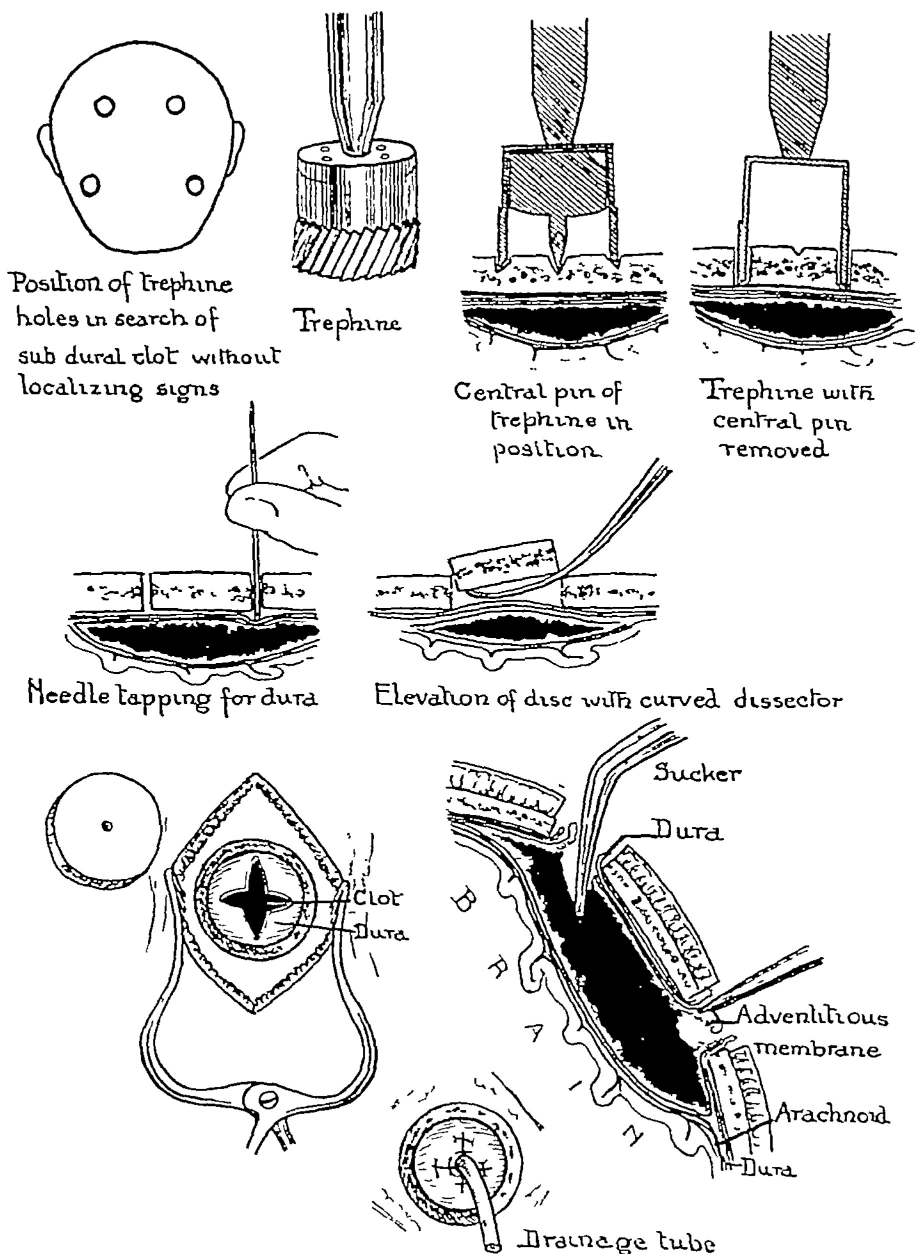


FIG 69

Evacuation and drainage of a chronic subdural hæmatoma

ally strap-like bands of thickened arachnoid deep in the wound have to be divided through an osteoplastic flap in order to let the brain expand.

Convalescence may be rapid but is often slow, causing anxiety even in those cases that will finally do well. Should the patient's

condition deteriorate at any time after operation, it is imperative to reopen the wound immediately to ascertain whether the clot has reformed or not. Also, the opposite side should be explored because subdural hæmatomata are often bilateral. When a clot is suspected but escapes detection by exploration, encephalography always discloses its position and size.

In those cases when the adventitious membrane is very thick, or when the clot is organised or calcified, it must be removed by dissection through the wide exposure of an osteoplastic flap.

Lumbar Puncture and Manometry.—In most cases, despite

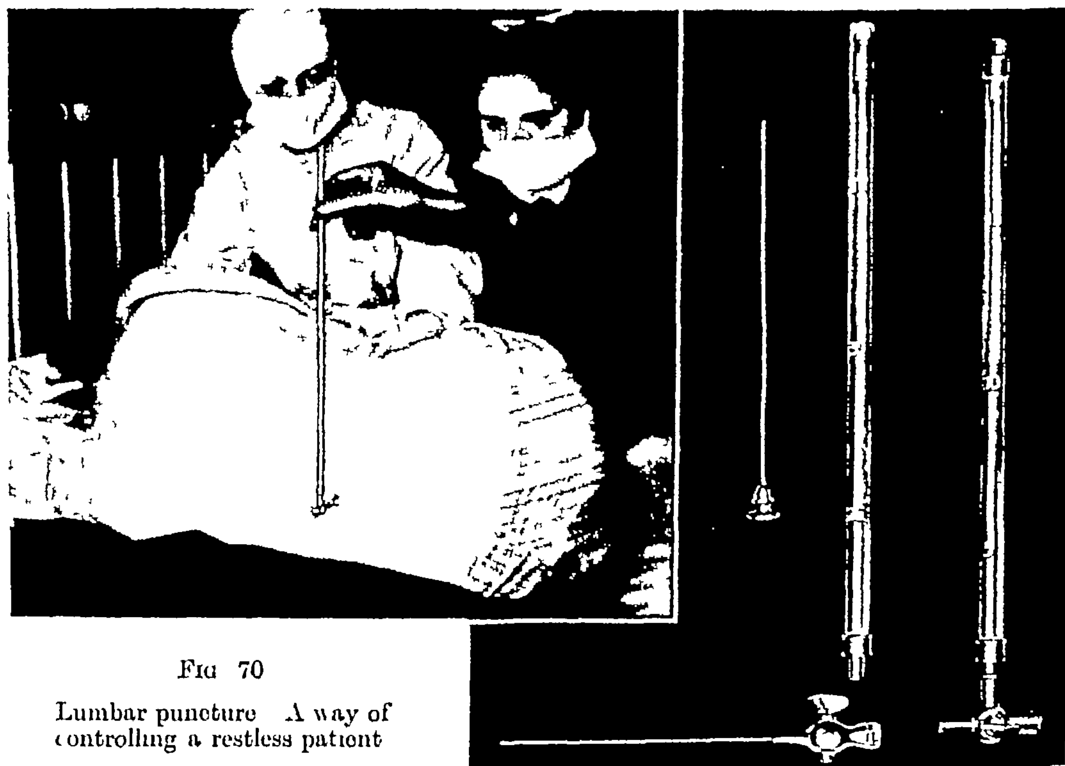


Fig. 70

Lumbar puncture. A way of controlling a restless patient

restlessness, a lumbar puncture can be done satisfactorily under local anæsthesia. A general anæsthetic must never be given to quiet a restless patient, as this will not only give a false pressure reading but is, in itself, dangerous. Usually two assistants are required to bring the patient into the necessary position and to hold him still while the puncture is made (Fig. 70). The patient is brought to the edge of his mattress under which a bed board has been placed, and his hip joints are fully flexed and his head bent forwards. A sterile towel is pushed beneath the patient's back by the operator and full aseptic precautions are taken. By means of a hypodermic needle the skin between the third and fourth lumbar spines is anæsthetised and the manometric needle is introduced exactly in the midline and angled backwards about

10°, so that it will travel a little towards the head. The resistance of the interspinous ligaments will be encountered and a jerk will be felt when the needle pierces the ligamentum subflavum and the dural envelope Absence of resistance means that the needle has left the middle line and may slide beside the theca and spike a posterior root. When this happens, or when a bony resistance is met, the needle must be immediately withdrawn and a fresh attempt made. A common mistake is to introduce the needle too deeply so that it passes through the far wall of the dural sac. This mistake can be avoided by withdrawing the stylet frequently to see if cerebrospinal fluid has been tapped and by rotating the needle so as to remove its bevel from the possible obstruction of a posterior root which may have floated against it. A dry tap indicates faulty technique.

When intracranial pressure is high, withdrawal of cerebrospinal fluid may lead to a tentorial or cerebellar pressure cone with resulting death. The danger of this complication, however, is outweighed by the importance of the information that only manometry can give.

As stated previously, pressure is measured in millimetres of cerebrospinal fluid, the normal range being between 50 and 150 mm. in the horizontal position.

Intravenous Transfusions of Hypertonic Solutions.—The apparatus is assembled as shown in Fig. 71.

The patient's arm is held by an assistant either along the edge of the bed or in abduction, according to the position which is most convenient to the surgeon, and the veins are made to distend by blowing up the arm band of the sphygmomanometer to venous pressure After the syringe has been partly filled with a 50 per cent. solution of sucrose through the side tube the needle is pushed into the vein, care being taken not to pierce its distal coat. The band of the sphygmomanometer is now allowed to deflate. By means of the stop-cock the syringe can be refilled without disturbing the needle and the injection continued until the required amount of fluid has been given. Care must be taken to keep the needle within the lumen of the vein, as leakage of hypertonic solutions in the subcutaneous tissues will cause a painful arm or may even lead to sloughing of the skin. If the tissues swell as the injection is being made, this means that the needle is not inside the lumen of the vein. When extravasation does occur the sucrose solution may be diluted by injecting sterile water into it, alternatively, the extravasated fluid may be let out through a small incision in the skin.

Sucrose, glucose and sodium chloride are most commonly used for hypertonic solutions; sucrose and glucose are used in 50

per cent. solutions and as much as 100 c.c. may be given at one injection; sodium chloride is used in 15 per cent. solutions and the maximum amount that may be given at a single injection is

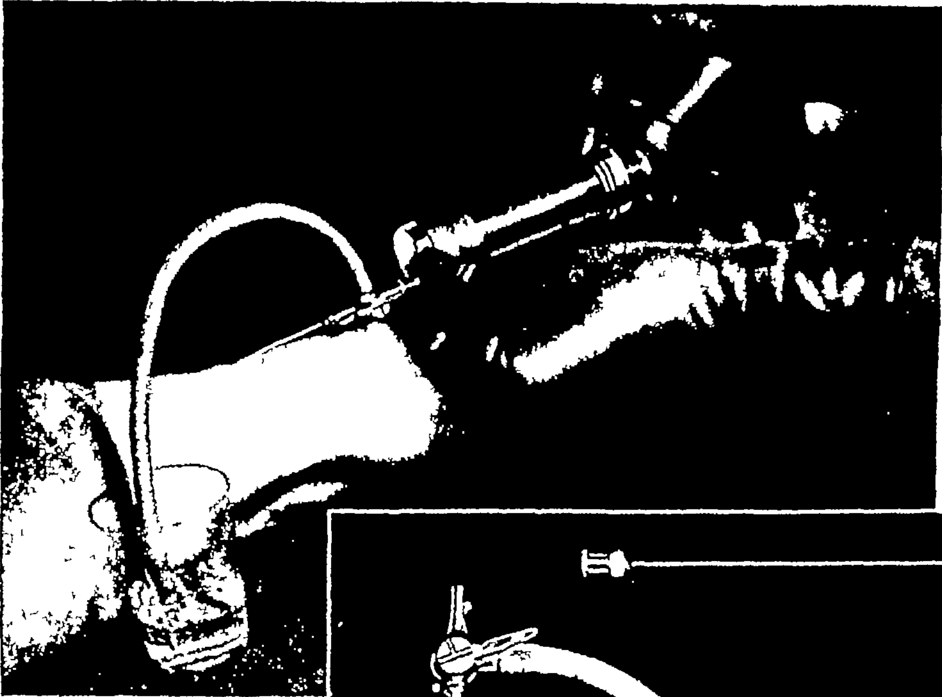
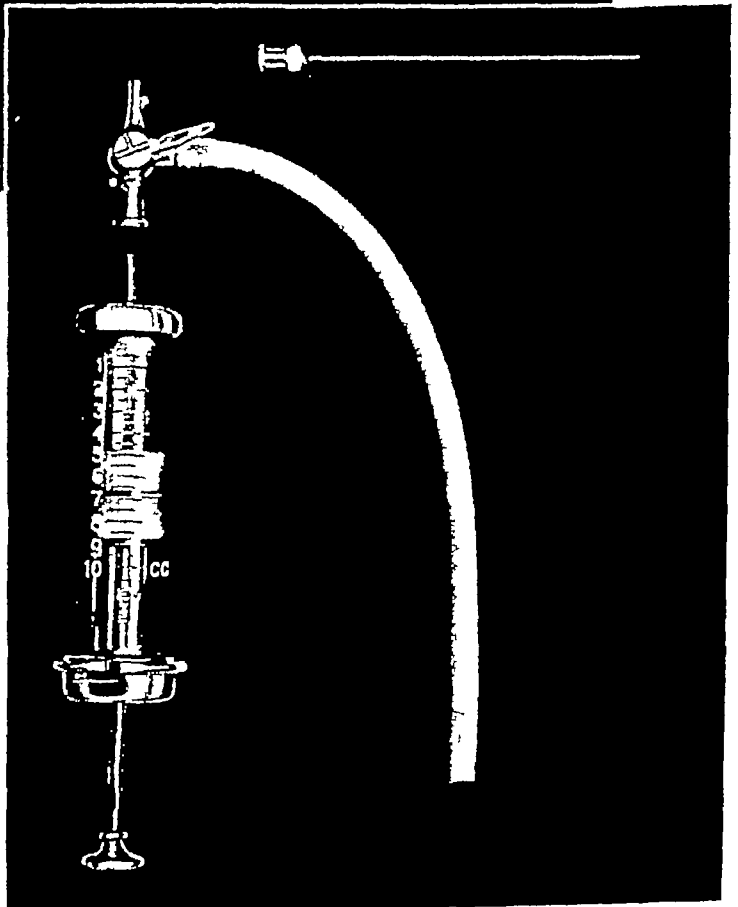


Fig 71

An intravenous transfusion



35 c.c. The advantage of sucrose over the other two substances is that its dehydrating action is more prolonged and that it is much less apt to cause secondary waves of cerebral œdema

The Stage of Convalescence.—Convalescence starts with the

return of consciousness and ends when the patient is able to get up and walk about without assistance. After a severe head injury three weeks may be allowed for this period.

The first week should be passed extremely quietly, reading or listening to the wireless being forbidden. When relatives are worried about a patient's mental state the importance of quiet should be fully explained to them, and they should be assured that improvement will almost certainly come in time. All anxiety, and in particular domestic worries, must be avoided. Tactful reassurance of the patient materially assists recovery. During the second week the patient is encouraged to move freely, his limbs are exercised and he should be occasionally lifted into an armchair when his bed is being made He may be allowed to read or listen to the wireless and talk with his fellow-patients if he wishes. A light but nourishing diet must be given and his bowels kept freely open with mild aperients. Headaches, dizziness, insomnia and depression are common symptoms; if they are belittled or overlooked, the confidence of the patient may easily be lost, since he will form the impression that the surgeon does not understand his case. On the other hand, if symptoms are given too much attention, a troublesome functional overlay may result. Careful judgment, therefore, is necessary on this score on the part of the doctor in charge.

Head pains may at first be exceedingly severe but will usually subside under correct treatment. Whether the head is kept low or raised on pillows depends entirely on the position that gives the most comfort. At this stage the cerebrospinal fluid pressure must be measured to guide treatment. As it is not often high, withdrawal of cerebrospinal fluid or intravenous dehydration will rarely give relief. When the pressure is subnormal, the head should be kept low and copious fluids given by mouth, so that a little more is taken than is actually needed to quench thirst. By these means an increase in the secretion of cerebrospinal fluid can be encouraged and the intracranial pressure brought back to normal. Aspirin and veganin may be used freely; small doses of bromides are soothing; and an adequate amount of sleep must be ensured, if need be by drugs such as nembutal, dial or medinal.

In all cases of compound injuries of the brain, and in closed injuries when epilepsy has occurred during the acute phases, sodium phenobarbitone ($\frac{1}{2}$ gr.) should be given night and morning to reduce the tendency to convulsive seizures.

The third week is an important period. Each day the patient is made to get out of bed for increasing periods; his muscles are toned up by gradual exercises until he can walk, and he is taught to bend and to stoop in an effort to restore his vasomotor control.

Period of Rehabilitation.—At this stage in his recovery the patient is allowed to leave hospital, but strict instructions must be given if he is to improve during the following weeks He must not be allowed to develop the habit of staying in bed in the mornings; he must have breakfast at the normal hour, and only then should he be allowed to rest if he finds it absolutely essential. Each day for a fortnight he should go or be taken into the fresh air, and should be firmly discouraged from huddling before the fire for long periods. Then at least two weeks should be spent at the seaside and the general health improved by a tonic of neurophosphates. Regular habits should be aimed at.

Return to work will now have to be considered. Unfortunately this problem is often complicated by the question of compensation. Immediate return to full or arduous duties is always undesirable, because the patient is apt to break down under the strain, and if he should do so his loss of confidence may initiate intractable neurasthenia. For example, it is most unwise to allow a middle-aged man who has been severely concussed to resume heavy manual labour or work amongst noisy machinery. On the other hand, a patient should be encouraged to adapt himself to his new physical state, and much economic waste could be avoided if really suitable employment could be found. The problems of rehabilitation and compensation will be discussed in Chapter VII.

CLOSED FRACTURES OF THE SKULL

Linear Fractures.—For undisplaced linear fractures of the closed type no surgical treatment is necessary. Within a few weeks a fracture line is filled with fibrous tissue, and for most purposes the protective efficiency of the skull is not materially impaired. Despite statements to the contrary, most linear fractures heal by bony union. This takes about twelve weeks in a child and from one to three years in an adult. When fractured surfaces are separated by more than a few millimetres fibrous union is the rule, but even in these cases isolated strands of bone often form and radiological evidence of fracture disappears. "Springing" of a suture is equivalent to a linear fracture and is often seen at the occipitoparietal junction Spicules of bone projecting from the inner table for more than ½ in. should be removed. This is done by cutting out the affected segment of bone with a trephine of suitable size The spicule is then removed by a chisel or nibbling forceps and the skull repaired by hammering the bony disc back into position It is unnecessary to keep a patient in bed for more than a few days because of a simple fracture of the skull. Indeed it is a mistake to call undue attention to its presence, owing to

the danger of encouraging some kind of troublesome functional overlay. Occasional observation, however, is necessary in order to make sure that an extradural clot does not form and compress the brain

The main difficulty in this type of case is the decision as to when a patient may take up his ordinary mode of life Recently I was asked to see a famous soccer internationalist who had received a linear fracture of his frontal bone without displacement. He was a vigorous forward, highly skilled in using his head to any kind of ball. A tribunal decided it was unwise to let him play again My opinion is that when radiology shows a fracture only as a fine line, a man may be allowed to play either association or rugby football at the end of a year whether the fracture heals by fibrous or bony union When a fracture is extensive and its opening is more than 3 mm. in width, it is unwise to give medical permission for such exercises, even though the danger of injury consequent on the weakness of the skull is exceedingly small It is, of course, quite justifiable to tell the patient that his skull is not seriously weakened and will withstand considerable violences without harm A linear fracture does not prevent a man from resuming his occupation even if he is a miner or quarryman, and certainly does not detract from the efficiency of a member of any of the Services.

Indentations.—Indentations may be pointed or rounded. They occur chiefly in children when the bones are thin and plastic and often are not associated with a demonstrable fracture. At birth, particularly following forceps delivery, rounded and large indentations are often seen in the skull Most of these disappear within the first few weeks as the child's brain develops, and need cause little anxiety The indications for surgical treatment are as follows :—

1 *At Birth*—Large indentations associated with signs of cerebral compression
2 *At Three Months.*—(*a*) Disfiguring indentations outside the hairline. (*b*) Any indentation over the motor cortex. (*c*) Indentations associated with symptoms such as epilepsy or obvious mental deficiency.
3 *At Three Years.*—All indentations irrespective of their position when more than 1 in in diameter

Methods—A small trephine hole is made at the periphery of the indentation. A suitably curved dissector is then passed through the operative opening and used as a lever to raise the depressed bone At the end of the operation the trephine disc is replaced so that a defect is not left in the skull When this method fails

owing to loss of plasticity of the bones, Dott has suggested that a hole should be made through the bone at the apex of the indentation and radial cuts made to the periphery. This allows each sector of bone so produced to be bent outwards at its base and moulded to the normal contour of the skull.

Depressed Fractures.—Depressed fractures of the closed type frequently involve large areas of bone and are caused by the head being struck over a broad surface. As in open fractures, they are often comminuted, although the affected bone is rarely broken into small fragments. Occasionally a complete bone is loosened at its suture lines and displaced not only inwards but sideways, so that one of its edges slides between the dura and intact skull.

Closed depressed fractures, even when extensive, are frequently unassociated with signs of local brain damage, and when a patient is conscious it is often difficult to decide whether their elevation by operation is necessary or not. When they are not causing obvious symptoms or signs and there is danger in raising them, as, for example, when they overlie the sagittal sinus, they are better left undisturbed. The decision to operate, of course, will be influenced by the age of the patient and his type of work. A weak skull in a schoolboy is obviously a serious disability, not only because it precludes him from playing games but it may seriously affect his chances of obtaining employment later in life. In such circumstances it is better to operate. On the other hand, when a man is living a sedentary life and has family responsibilities, it is wiser not to operate.

The specific indications for operative treatment are as follows:—

1. When a patient is unconscious and thought to be suffering from cerebral compression.
2. When there are signs of underlying brain damage, such as hemiplegia or aphasia, or when a patient has persistent symptoms of headache and giddiness.
3. When a fragment of bone is thought to have pierced the dura. This judgment depends on the shape of the bony fragment, on its angle of tilt and on the amount of depression. Fragments depressed for more than ½ in., those lying end on, or those obviously spiculated should be elevated.
4. Cosmetic considerations are important not only in women but also in men. Depression and loss of confidence may often be traced to an inferiority complex caused by brooding over a physical disability which is noticeable to other people. This functional complication should never be overlooked, as it can often be corrected by repairs of the skull.

The raising of a large fragment of bone is not a simple operation and cannot be effected by leverage through a trephine hole. It necessitates exposure of the whole of the depressed fragment by means of a suitably designed skin flap. The decision as to the type of operation necessary depends on the findings after the skin flap has been raised.

When the depressed fragments are loose and move easily, all that is required is to lift them and to arrange them in position. They may be anchored by sutures passed through the pericranium covering them. This, however, is generally unnecessary, as they are usually held firmly enough in position between the dura and scalp as the skin flap is closed. Completely loose fragments without blood supply will readily consolidate, and no misgivings need be entertained on this score Also, tight fitting, as in grafting in other bones, is not essential or even important.

A block resection of depressed fragments is necessary when they are tightly interlocked and cannot be disentangled As described in the next chapter, a block of bone is removed in these cases and the displaced fragments hammered into position The remoulded block of bone is then replaced and anchored by means of silk or fine wire sutures passed through drill holes made in suitable and corresponding positions in the block and surrounding skull Often suture of the pericranium of the graft to that of the surrounding skull will suffice to keep the graft in position until it consolidates. In old-standing depressed fractures, when the bony fragments have united by fibrous or bony union, the bone after block resection may be reversed so that its inner surface faces outwards if the contour of the skull will allow When this is not possible, the fragment has to be broken up by the best means available at the time. No set operation can be described for these cases, as the conditions are so variable. Each case, therefore, has to be judged on its merits, the actual corrective manœuvre depending on the surgeon's skill and ingenuity.

DEFECTS IN THE SKULL

The most common cause of defects in the skull is the operative removal of bone fragments in the débridement of compound depressed fractures Less frequently, defects are the result of explorations and decompressions made for the evacuation of extradural clots or for the relief of pressure in cerebral œdema. Loss of bone in road and industrial accidents caused by an injuring force is extremely rare and is no more common in air-raid casualties. It does, of course, occur more often in gunshot wounds, but many of these are fatal. In children, skull defects occasionally result

from destruction of an epiphysis by injury of the closed type. This leads to disappearance of bone in a wide area, as shown in Chapter II, Fig. 23, and is known as traumatic malacia.

The disability caused by a calvarial defect is not necessarily serious and depends on its size, position and the part it plays in the production of post-traumatic epilepsy. A defect naturally is much more serious when the underlying dura is open and the brain is adherent to the scalp than when the dura is intact, as drag on the cerebral cortex is so prone to cause headaches, dizziness or epilepsy. It is often difficult to decide, particularly in the absence of symptoms, whether or not a repair of the skull is necessary, and it will be helpful to keep the objects of treatment clearly in mind. These are fivefold :—

(i) *For the Cure or Prevention of Traumatic Epilepsy.*—As the cause and treatment of traumatic epilepsy will be discussed in a later chapter, all that need be said here is that, according to circumstances, adequate repair of the skull may be an important factor in its cure.

(ii) *For Protection of the Brain.*—In young people, repair of a defect in the skull is necessary if their normal activities such as the playing of games are not to be restricted. This is an important consideration, and parents will usually give consent to an operation when the arguments for and against repair are presented to them. A weak skull is obviously a serious disability in miners or industrial workers whose heads are liable to be knocked on account of the nature of their work. Correctly fitting protective helmets or splints will give adequate protection under these conditions, but often a workman refuses to wear them, or finds them uncomfortable or too hot to be tolerated for long periods. When objections to external protective methods are made, operative repair of a defect is fully justified and usually gives excellent results. In members of the Services a calvarial defect usually leads to discharge or to regrading to a lower physical category. This is often unnecessary, since a simple bone graft would make the man concerned just as efficient as before.

(iii) *For Cosmetic Purposes.*—Disfigurement cannot be measured by scientific standards. It is purely a matter of personal opinion, and the only point of importance is the opinion of the patient himself or, in the case of a child, that of the parents. If the appearance of a defect is worrying to a patient, then it should be repaired wherever or however small it may be. Very few people will overlook the

unsightly deformity caused by the loss of a supra-orbital ridge, particularly if the brain slips forward and bulges over the eye.

(iv) *For Relief of Giddiness and other Symptoms consequent on Instability of the Cerebral Circulation.*

(v) *For Psychological Reasons*

TREATMENT

General Considerations.—Repair of a calvarial defect must never be considered until a wound is free from infection and the skin soundly healed over it In the presence of infection a graft will not consolidate and will, in fact, aggravate inflammatory processes by acting as a foreign body and increase suppuration until it is discharged from the wound. Unfortunately skin may heal over tissues which are potentially infected. On those occasions, therefore, when the original operation was not done by oneself, it is essential to know how thoroughly débridement was carried out in the first instance before deciding to reopen a wound. An unhealthy-looking scar, repeatedly scabbing in spite of treatment, is suggestive of infection and must be regarded as such. After a few months, radiography often will give valuable information regarding the presence or absence of infection, since in a clean wound the margins of the bone will be sclerosed, whereas when infection is active the bony edges may be fluffy or sequestra may show.

As a working rule it is wiser to wait for three months, even in favourable cases, before operative measures are considered, and this period should be even longer in doubtful cases When operative repair of a defect is not advisable and protection is necessary, a metal splint moulded to the shape of the head and held in position by suitable straps is the usual method adopted by instrument makers Such metal splints are often uncomfortable, and I have found that a felt pad is more useful than a metal plate This can be sewn into the cap or hat, thereby avoiding the necessity of tight strapping to hold it in position

General Operative Technique.—A skin incision is designed to expose the whole of the calvarial defect concerned including the tissues beyond its margins for a distance of at least ½ in. Scars made at a previous operation will necessarily influence the shape of an incision In particular a semicircular flap must be so oriented that a scar does not run across its base, otherwise its blood supply may be inadequate. On the forehead an incision running vertically in the middle line will heal with the minimum of obvious scarring. A frontal defect may be exposed by continuing the upper end of a vertical incision outwards and downwards

into the temporal fossa within the hair-line, and by turning the skin flap thus produced forwards and outwards (Fig 72). When a stellate scar is present every effort must be made to avoid opening all three limbs, because healing at the apex is apt to be delayed if this is done. Usually all that is necessary to give an adequate exposure is to open two limbs, which may be prolonged if circumstances demand.



Fig 72

This type of incision for exposure of a defect in the frontal bone leaves a very inconspicuous scar

After the skin has been reflected the pericranium is usually found to be densely adherent to the dura over the edges of the bony defect. When this occurs, an incision through the pericranium at the periphery of the bony opening will be necessary before it is possible to separate the dura from the bone. Such separation often causes considerable bleeding, and great care must be taken to have the dissector under control when this manœuvre is carried out, otherwise the dura may be torn and the brain lacerated. The object of freeing the dura is to ensure that grafts fit snugly into the defect and to enable holes to be drilled into the bone if the graft has to be sutured into position by this means.

Methods of repairing a Bony Defect.—The older method of insinuating a piece of tin-foil or perforated celluloid plate between the dura and bone has been superseded by the use of bone grafts which may be taken from (1) the external table of intact skull, (2) the ilium, (3) the tibia or (4) the ribs Autogenous grafts are the best, but in children bone may be taken from the mother or from a donor of the same blood group

Grafts taken from the External Table of the Skull—A piece of sterile lint or gutta-percha tissue accurately fitting the bone defect is cut out and placed over intact skull which, according to the size of the graft required and to the vascular requirements of the scalp, may or may not need to be exposed through a separate incision An incision is then made through the pericranium ½ in. outside the boundaries of the pattern, and the membrane separated inwards with a rongeur for the same distance so that a flange of pericranium is produced By means of a circular

saw the external table is cut in the shape of the pattern and the graft removed by means of chiselling through the diploe. The graft is then fitted into the defect and its flange of periosteum stitched to that of the surrounding skull. One of the difficulties of this method is that the graft is apt to splinter when being lifted. Also, it is inapplicable to children, in whom the diploic cavity is absent or imperfectly formed.

Rib Grafts.—The advantages of rib grafts are that they are easily taken and readily moulded to the contour of the skull. When this method has been decided upon, the lower ribs are exposed in the mid-axillary line and lengths of bone removed subperiosteally according to the size of the defect to be repaired. After removal, the rib is split longitudinally along its broad plane so that two flat grafts are produced. By suitable transverse cuts the bone may be weakened and so bent that it fits the contour of the surrounding skull. Grafts should always be arranged so that their smooth surface faces the dura.

An alternative method of cutting a graft is to make incisions through the fibroperiosteum at the upper and lower edges of the rib instead of centrally, as in purely subperiosteal resections. Thus one half of the graft will have a fibrous covering. It is dangerous to attempt to separate the deep surface of the rib extraperiosteally because of the danger of rupturing intercostal vessels before they are sufficiently exposed to be ligatured.

A subperiosteal graft, *i.e.*, a graft without a fibrous covering, may be dovetailed into the diploic cavity at the margins of a defect after the tables of the skull have been prised open by suitable chiselling Usually it is better to sew a graft in position by silk or wire sutures after corresponding holes have been drilled through its ends and through the skull. When holes are being made through the skull, the brain must be protected from the drill by means of a special pair of forceps (Fig. 73) that grip the bone between its two jaws, the upper of which is perforated to allow the drill to pass. When this instrument is not available a flat metal retractor may be slipped between the skull and the dura to protect the brain.

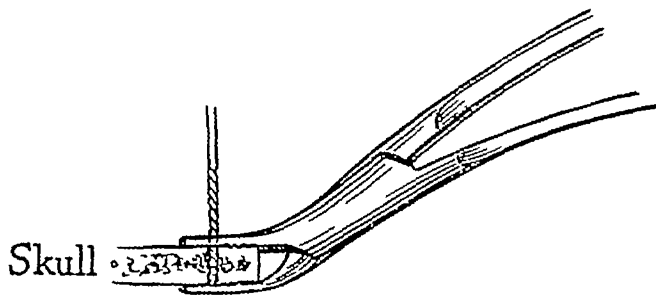


FIG 73
Adson's drill forceps

Grafts taken from the Tibia.—According to the area of bone required to repair a defect, a length of the superficial surface of

the tibia from which it is proposed to take the graft is exposed through a straight or curved incision. If necessary manipulations are not to be impeded, a generous exposure of the tibial surface should be made. After the periosteum has been incised along the extreme anterior and antero-internal edges and separated cen-

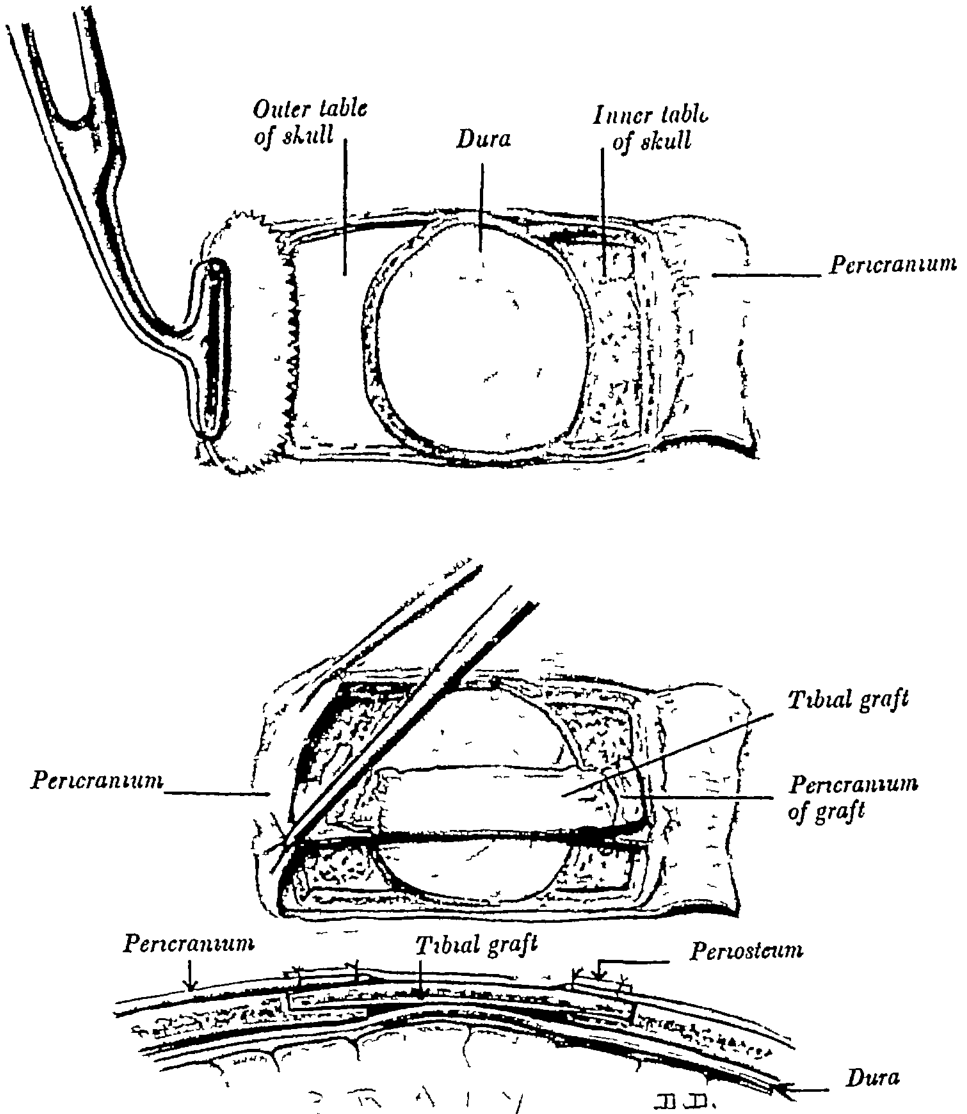


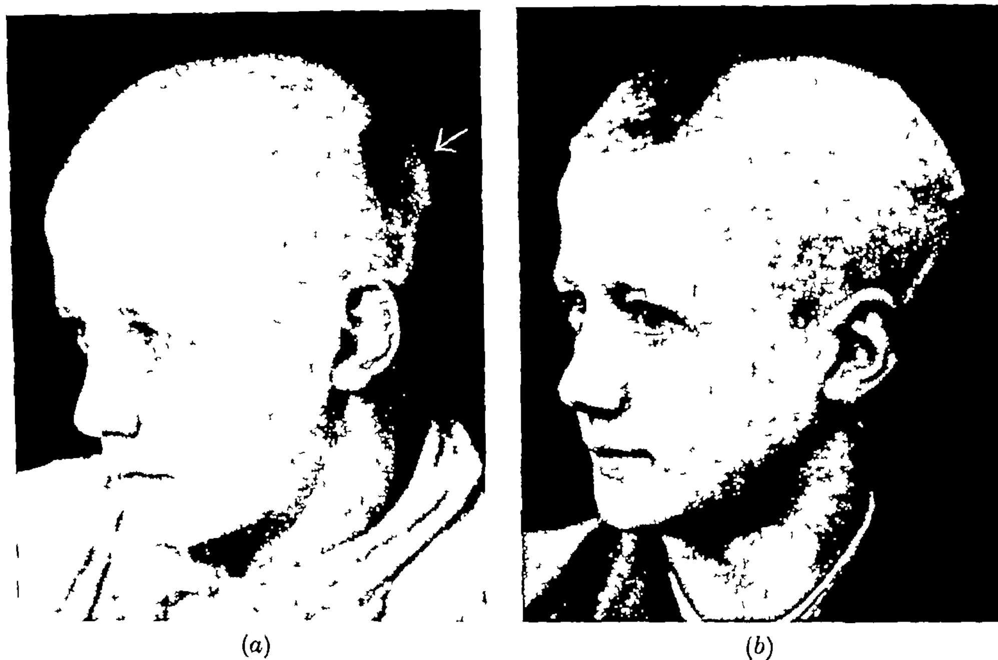
FIG 74

A tibial inlay graft for repair of a calvarial defect

trally, longitudinal cuts are made in the bone with a curved hand-saw, just within the line of the periosteal incision, to a depth of 3 mm. Then periosteum and bone are cut transversely and a shaving of tibia 2 to 3 mm thick is taken between the saw cuts by means of a wide chisel, the length of the graft depending on the method to be used for fixing it in position

Fixation by the Bone-shelf Method—After reflection of the

scalp and separation of the dura, a decision has to be made along which axis of the defect the laying of the grafts will give the best cosmetic results Usually grafts are best lain along the flatter of two curves. Whether this is in the coronal or sagittal plane depends on which part of the skull the operation is to be done. When the axis has been chosen, a sunken square or rectangle, as shown in Fig 74, is made to receive the grafts by removing the outer table of the skull. Removal of the outer table may conveniently be done by means of a curved hand-saw and a chisel. The tibial grafts are then placed in position as an inlay and their covering periosteum sewn to that of the surrounding skull.



(*a*) (*b*)

FIG 75

a, A defect in the parietal bone *b*, The same case after repair of the defect by means of a tibial graft placed between the bone and pericranium

Fixation of Subpericranial Implantations—This method has been used extensively by McGill and myself, and we find that its great advantage is its technical simplicity. Also, final consolidation of the graft by this method is as good as any other. Cosmetically it is satisfactory (Fig 75).

The bone grafts from the tibia are cut as described above, and of such length that they will overlap the edges of the defect by $\frac{1}{4}$ in. at each extremity of the axis along which the grafts are to be placed. The pericranium is then separated from the skull but not completely detached, and the periosteum at the ends of the grafts is lifted as a flap The denuded ends of the graft are then slipped beneath the curtain of pericranium and the grafts sewn into position, as shown in the accompanying diagram (Fig

76) The scalp is sutured in the usual way, drainage being unnecessary. The patient may be allowed to get up in forty-eight

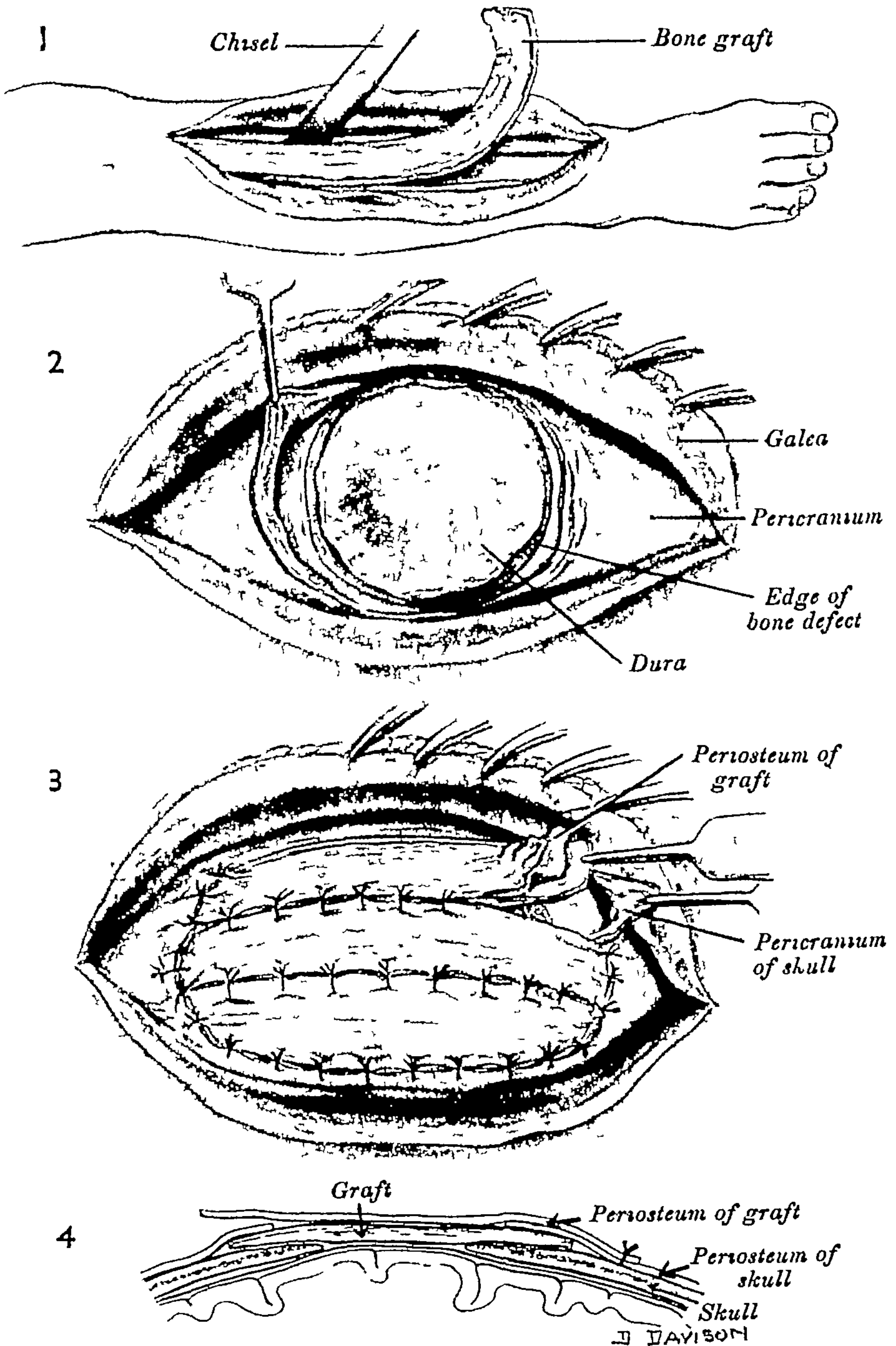


FIG 76
Repair of a calvarial defect by a tibial graft placed between the bone and pericranium

hours, and after six weeks no protective covering on the head is necessary.

CHAPTER V

OPEN OR COMPOUND WOUNDS OF THE HEAD

FIRST AID AND TRANSPORT

ROAD Accidents.—In road accidents shock and bleeding from the scalp are the only urgencies which a medical man is likely to encounter and have to treat when a patient has been injured about his head. Other types of injury are either of such severity that they are beyond the aid of medical measures or can wait without deterioration for treatment until adequate surgical facilities are available. Owing to shock, bleeding often stops spontaneously, but on those occasions when it does not, immediate action must be taken if excessive amounts of blood are not to be lost. As it is only by chance that surgical material may be available, first-aid ambulance methods are usually necessary to still bleeding by the application of pressure. This may be done most conveniently by means of the two-handkerchief method (Fig. 77). First a clean piece of linen is put over the wound and then a second handkerchief or scarf folded into a triangle, with its base placed across the forehead, is tied in position by knotting its lateral tails below the external occipital protuberance. By pulling on the apical tail, pressure can be applied over the first handkerchief and the bleeding stopped. Such bandaging, of course, must be done judiciously, otherwise loose fragments of bone may be driven inwards to compress or lacerate the brain.

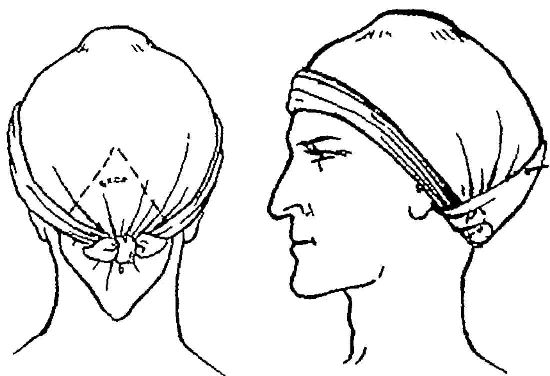


FIG 77

The two-handkerchief method of controlling bleeding in the scalp until surgical facilities are available

Air-raid Casualties.—During and following air raids little can be done for the unconscious with closed head injuries, and such cases should be removed to hospital as soon as possible.

Surgical outposts, as far as can be seen at the moment, are mainly concerned with open wounds, and here the main problems

are. (1) The treatment of shock, pain, fear and restlessness; (2) the prevention of sepsis, and (3) decisions regarding transport[1] A simple wound of the scalp or one associated with a clean linear fracture may be sutured at once, if reasonable aseptic precautions can be taken such as shaving of the scalp. A warning, however, is necessary not to overlook the possibility of a severe cerebral injury in the presence of a simple scalp wound When a lacerated scalp is associated with a cerebral injury it is better to leave the wound open, apart possibly from a few tension sutures to control bleeding, because in these circumstances if a wound has been carefully sutured, the surgeon at the receiving hospital may be tempted to leave it intact and not to explore as possibly he ought the depths of the wound. In depressed fractures it is wise always to be conservative and delay radical treatment until adequate surgical facilities are available, since the simple lifting of a depressed fragment may start uncontrollable bleeding. Also, when the dura mater is opened, manipulations in the absence of perfect aseptic precautions will almost certainly lead to spreading encephalitis Bleeding can usually be stopped by means of light pressure applied over a sterile dressing On no account must the source of bleeding be sought deeper than the scalp, as this is almost certain to increase the hæmorrhage and disseminate infection. Bleeding from the edge of the scalp may be controlled by hæmostats or preferably by pulling the wound loosely together with sutures. To attempt to repair a complicated wound without adequate assistance and full surgical facilities is a serious error in judgment

Pain, fear and restlessness may necessitate the administration of small doses of morphia, but drugs are better withheld if there is no delay in transport. Prevention of sepsis is synonymous with adequate excision, wherever this may be done, and it is very wrong to regard chemotherapy as the panacea against infection Shock is treated by rest and warmth; blood transfusion is necessary for hæmorrhage.

From the point of view of transport, the injured may be grouped thus:—

(i) The moribund and those seriously ill.
(ii) The shocked and the exsanguinated.
(iii) The violently restless
(iv) Those with minor injuries.

In times of stress it is permissible in severe cases to give precedence to those with the better prognosis and to keep the

[1] Ministry of Health Emergency Medical Service "Memorandum on the Treatment of Head Injuries" E M S, Gen, 280

moribund behind, as the latter are likely to die before they reach the main hospital In any case little can be done for them surgically when they have received severe intrinsic injuries to the brain

Patients with minor injuries may be allowed to go home or may be sent for a short period to rest in neighbouring houses.

Whenever a wound has been sutured, the details of the exploratory findings must be stated quite clearly in the patient's notes, and it is essential to make it clear whether a satisfactory repair has been made or whether the wound must be reopened for more extensive excision at the main hospital. Moreover, it is always useful for a surgeon to know whether the dura mater has been lacerated, since tears of this membrane are in need of more urgent treatment than injuries confined to the scalp or bone. As in closed head injuries, neurological details of the early phases of the injury are useful in guiding subsequent treatment.

General Considerations.—The principles of diagnosis and treatment in closed injuries of the head apply equally to open wounds of the vault. A detailed neurological examination must therefore be made in all cases, however trivial a superficial wound may be, so as to determine whether an injury to the brain has occurred, and if so to localise it and to diagnose its nature Without this information it is impossible to treat a case correctly, since local appearances in a wound may give no indication as to the damage done deeper in For example, a linear fracture may have to be trepanned if there are neurological signs of an underlying extradural or subdural hæmorrhage

Treatment, though primarily directed to excision and repair of wounds, is also designed to keep intracranial pressure within normal limits in the manner described in the previous chapter

Wounds of all types may be classified according to their depths,[1,2] thus :—

(i) Wounds confined to the scalp.
(ii) Wounds associated with linear fractures of the skull.
(iii) Compound depressed fractures with intact dura mater.
(iv) Compound depressed fractures with torn dura mater.
(v) Indriven fragments and retained missiles.
(vi) Wounds opening into the mastoid or paranasal air sinuses.

It is important to know that the dura acts as a very efficient barrier to the spread of infection into the brain tissue or meningeal spaces. In fact a classification of wounds could be made on the

[1] Cushing, H ' Study of a Series of Wounds involving the Brain and its Enveloping Structures " *Brit Jour Surg*, 1917-18, **5**, 558

[2] Jefferson, G " War Wounds and Air-raid Casualties War Wounds of the Head—I " *Brit Med Jour*, 1939, **2**, 347

basis of dural integrity, since this is of the greatest prognostic value. When dura is intact a patient is very unlikely to die from infection if a wound is carefully excised. On the other hand, when the dura is torn there is always grave danger of encephalitis and meningitis, however carefully or early débridement is carried out.

Pre-operative Considerations.—A wound may be explored pre-operatively to determine its depth in order to get some kind of guidance to the magnitude of operation that later may be necessary, and this is best done with a sterile finger, since probing with a metal instrument is more dangerous as regards dissemination of infection and less likely to give satisfactory information. The main objection to pre-operative exploration is that troublesome bleeding may be started which is not easy to stop by simple means.

Associated injuries are often present, and whether their treatment is more urgent than the head wound must be decided in each case. Usually this decision is not difficult. Although the ideal time to operate on a patient with a compound wound of the head is within twelve hours of the receipt of injury, many patients with multiple injuries may safely be left for eighteen or even twenty-four hours from the point of view of infection

In any case it is most unwise to operate on a patient before he has recovered from primary shock, and certainly many cases are unnecessarily lost through failure to observe surgical first principles When in doubt about the ability of a patient to withstand an extensive operation on the head, it is better to wait than to be in a hurry. In one case I delayed operation for two and a half days with gratifying results

X-rays.—A pre-operative X-ray examination of the skull must never be omitted, as this gives direct evidence of what otherwise must often be conjectural. It will show the extent of a bony injury, the size of a depression, the number and exact site of indriven bony fragments, and the presence or absence of radio-opaque missiles, such as bullets or pieces of shrapnel. In air-raid casualties radio opaque material is usually matted in the hair, and therefore Harvey Jackson,[1] with much reason, has suggested that radiographic examination is better done after the head has been shaved

Choice of Anæsthesia.—Local anæsthesia is the method of choice in the treatment of a compound wound of the head. It is safer than other methods from the patient's point of view, and as a surgeon becomes accustomed to its use he will soon realise how much easier it makes the actual operating. By this means intracranial pressure is minimised and troublesome venous oozing avoided. Moreover, when the dura mater is torn a general

[1] Jackson, H Paper read at the meeting of the Neurosurgical Society, Oxford, July 1941

anæsthetic is dangerous, because it invariably causes a rise of some degree in intracranial pressure, which may initiate or increase an existing cerebral hernia.

Novocaine should not be injected into or near the edges of a wound but some distance away, in the form of a ring to enclose the proposed operative field. This procedure not only keeps the anæsthetic clear of damaged and infected tissues but also allows easy extension of a wound if this is found necessary.

General anæsthesia[1] is indicated when a patient is restless and unco-operative and when the scalp is so extensively lacerated that it is impossible to shave without causing severe pain. It may also be necessary if other injuries are to be treated at the same time as the head wound is operated on When a patient is fully conscious and has recovered from shock, a general anæsthetic may always be given, provided that the dura mater is intact.

Preparation of the Scalp.—Unless a wound is small and obviously superficial the whole scalp should be shaved and the skin cleaned in the usual way Local shavings are to be condemned, since the adjacent hair becomes matted with blood and acts as a breeding ground for bacteria Moreover, difficulties may arise at the time of the operation if the operative field has to be enlarged.

In air-raid casualties shaving of the head has proved to be an extremely difficult and lengthy manœuvre, because grit and dirt blown into the hair and skin by the explosion blunt the razor edge after a few sweeps across the scalp. The method I have found most useful is to cut the hair short with scissors, comb it, then to scrub it with a sterile nail-brush and soap and water and finally to shave it. Botterell has shown that a safety-razor blade gripped in the jaws of an artery forceps is an excellent way of always having a sharp and efficient tool at hand. Preparation of a head is best done in a bathroom where the patient can be thoroughly washed with warm water all over the body, because large quantities of dirt often collect in the axillæ and groins and cause discomfort

THE SCALP

Applied Anatomy.—The calvarium or vault of the skull has five coverings and each one is surgically important. From within outwards these are :—

(i) The pericranium.
(ii) Loose areolar tissue.
(iii) Galea aponeurotica.
(iv) Superficial fascia.
(v) Skin.

[1] Brennan, H J ' Gas-oxygen and Cerebral Congestion " *Lancet*, Feb 1938, 1, 315.

The pericranium (Fig. 78) is firmly attached to suture lines but readily separable elsewhere, and therefore hæmorrhages in the subpericranial layer remain confined within the boundaries of a single bone area. Examples of this type of bleeding occur

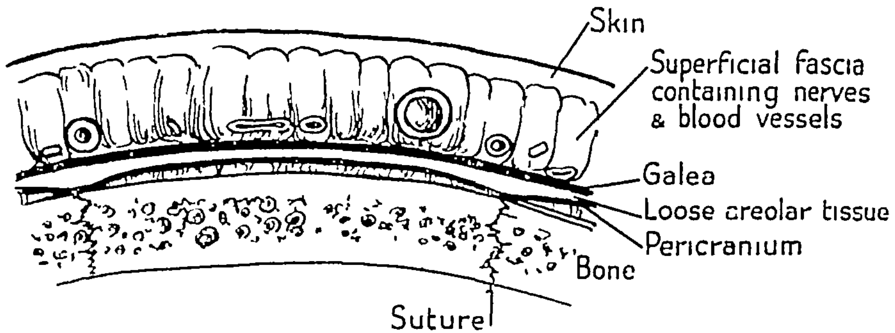


Fig 78
Cross section of the scalp

in birth injuries when so-called cephalhæmatomata are to be seen standing up like mounds on the head, in the region of the parietal bones.

The Layer of Loose Areolar Tissue.—The looseness of the attachment of the areolar tissue, both to the galea and to the pericranium, explains why the skin can move freely over the surface of the skull. This is of great surgical importance, since it allows closure of a wound even after considerable areas of tissue have been lost. Another point of surgical importance is that infection can spread rapidly with little hindrance along the areolar layer If, therefore, serious complications are to be avoided, radical and early drainage of suppurative processes in the subgaleal space is essential

The galea aponeurotica or the tendon of the occipito-frontalis muscle is attached directly or indirectly to the whole circumference of the base of the calvarium, thus forming a fibrous tissue envelope for the skull A wound involving only the skin does not gape, whereas, when the galea has also been incised, gaping does occur. The galea is of particular surgical value in the repair of extensive wounds of the scalp because it is tough and may be sewn together with buried sutures, which allows skin sutures to be removed in a few days' time. This minimises the dangerous possibility of stitch abscesses and also to a large extent eliminates disfiguring scars

The Superficial Fascia or Subcutaneous Tissue.—This tissue binds the skin proper firmly to the underlying galea and therefore no movement is possible between the more superficial layers of the scalp. Also, it is in the layer of superficial fascia that the main nerves and blood vessels are found, and owing to the fibrous

nature of the tissue normal retraction and contraction of severed blood vessels is prevented, thus accounting for the profusion of bleeding when the scalp is cut

The skin is so richly supplied with blood vessels that even in a small wound of the scalp a patient may lose a considerable amount of blood in a very short time. In view of this, therefore, very great care must be taken when operating on the head to have the blood vessels under control by digital compression before cutting them. The advantage of a rich blood supply is that wounds heal well and that flaps of tissue with a small base will live. The richness of the scalp's nerve supply means that cuts of the scalp and operations on the scalp cause considerable shock if the skin incision is not blocked with local anæsthesia.

The Nerves of the Scalp (Fig. 79).—The trigeminal nerve

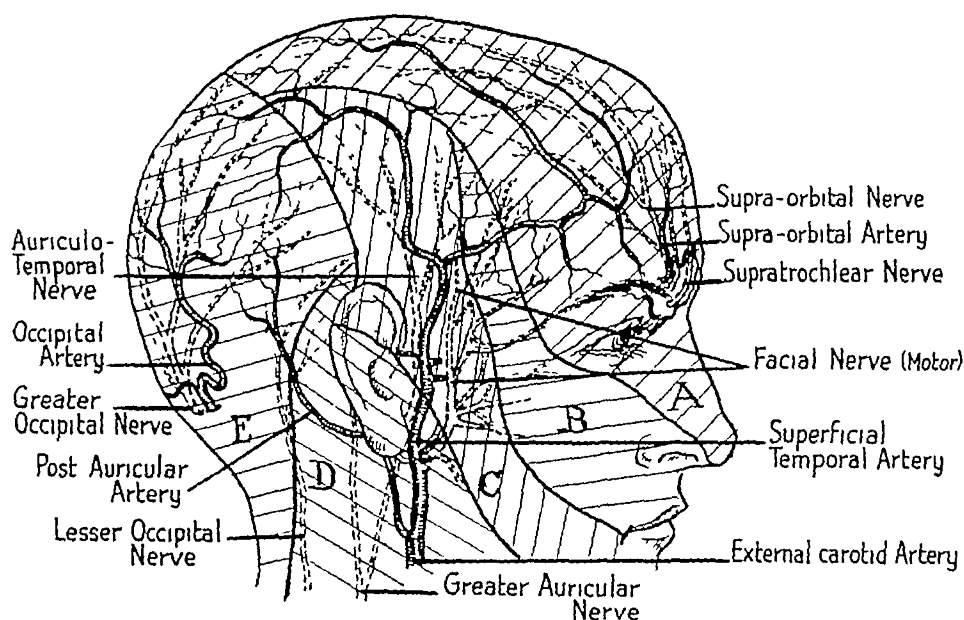


FIG 79

The blood vessels and nerve supply of the scalp

A, Area supplied by ophthalmic division of the trigeminus, B, area supplied by the second division of the trigeminus, C, area supplied by the third division of the trigeminus, D, area supplied by the anterior divisions of the upper cervical plexus, E, area supplied by the posterior divisions of the upper cervical plexus

supplies the anterior two-thirds of the scalp and the cervical plexus the posterior third.

(*a*) *Branches of the Trigeminal Nerve*—

Division 1.— (i) Supra-trochlear.
(ii) Supra-orbital.
Division 2.—(iii) Zygomatico-temporal.
Division 3.—(iv) Auriculo-temporal.

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(*b*) *Branches of the Upper Cervical Plexus*—

(i) The great auricular.
(ii) The lesser occipital
(iii) The great occipital.

The Blood Vessels of the Scalp.—The blood vessels on each side of the scalp freely anastomose. Except in the region of the forehead, however, anastomosis is poor across the middle line. Where possible incisions in the midline on the top and at the back of the head should be avoided. Also, as the arteries of the scalp run from below upwards, incisions in the scalp should be made vertically and not transversely. Skin flaps should be turned downwards and not upwards.

The arteries of the scalp originate from :

(*a*) Internal Carotid Artery { Frontal. / Supra-orbital.

(*b*) External Carotid Artery { Superficial Temporal. / Posterior Auricular. / Occipital.

Veins.—The frontal and supra-orbital veins drain via the ophthalmic veins into the cavernous sinus.

The temporal and posterior auricular veins drain into the internal jugular veins.

The occipital veins drain into —

(*a*) The deep cervical veins.
(*b*) The vertebral veins.

The veins of the scalp are freely connected with the parasaggital, lateral and cavernous venous sinuses through so-called emissary veins. Not only, therefore, does bleeding occur from the bone when the flap is lifted because of these communicating channels, but also infection from the scalp can easily gain access into the intracranial cavity by means of thrombophlebitic processes

Lymphatics of Scalp.—

(i) The occipital region drains into glands on the insertion of trapezius muscle at the back of the head.
(ii) The parietal region drains into glands behind the ear over the insertion of sternomastoid muscle.
(iii) The anterior region of the scalp drains into the glands in front of the ear over the parotid gland.

Infection of the scalp always leads to prolonged suppuration. Therefore if valuable time is not to be wasted, both for the patient and for the hospital concerned, every wound, however

trivial, must be carefully cleaned, trimmed and accurately sutured (Fig 80) The exact details of an excision naturally will depend on the size, shape, raggedness or degree of contamination of a wound The main principle to be observed in débridement is that edges of skin should be cut away sparingly with a sharp scalpel and foreign bodies removed from the depths of the wound.

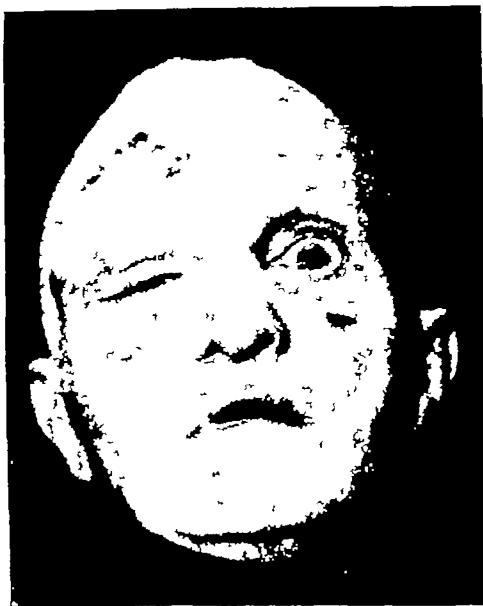


Fig 80

Cellulitis of the scalp is often the result of an inadequate excision of a simple wound.

Though the skin is firmly attached to the galea, the galea moves freely over the pericranium, and thus with suitably placed incisions the edges of a wound may be mobilised and freely retracted if a wider exposure of deeper tissues is found necessary Alternatively, by suitable incisions and sliding of the scalp, a wound may be drawn together even if there has been considerable loss of tissue. The conversion of a defect into a long ellipse is a most useful method of effecting closure when a wound is not very ragged and when loss of tissue has not been great The S-shaped incision and the triradiate incision of Cushing for facilitating exposure or closure are the most suitable when a wound is irregular and much tissue has been lost. Without loss of tissue they should never be used. Semicircular skin flaps may be necessary to avoid continuing incisions on to the face or into the neck, but it is the general belief that they should be used as rarely as possible, since, if they do not heal by first intention, they will pull apart and gape to a much greater extent than a triradiate incision would have done The advantage of a flap is that an adequate exposure of the deeper tissues can always be obtained. It is a method I commonly use myself Dott [1] also advocates the use of this method.

Lateral incisions that are allowed to gape in order to facilitate sliding of the edges of the original wound to cover an exposed area of brain are sometimes necessary, but at best are unsatisfactory

Incisions for the better-known procedures for plastic repair of the scalp are shown in Fig 81 On those occasions when tissue has been lost, forcible apposition of the skin by mattress sutures is useless, because the margins of the flaps will certainly slough

[1] Dott, N M "Surgery of Modern Warfare," 1941, 67

and the wound break down. The maximum tension permissible in a wound difficult to close can be judged by buried sutures. and it may be accepted as a working rule that if the skin can be

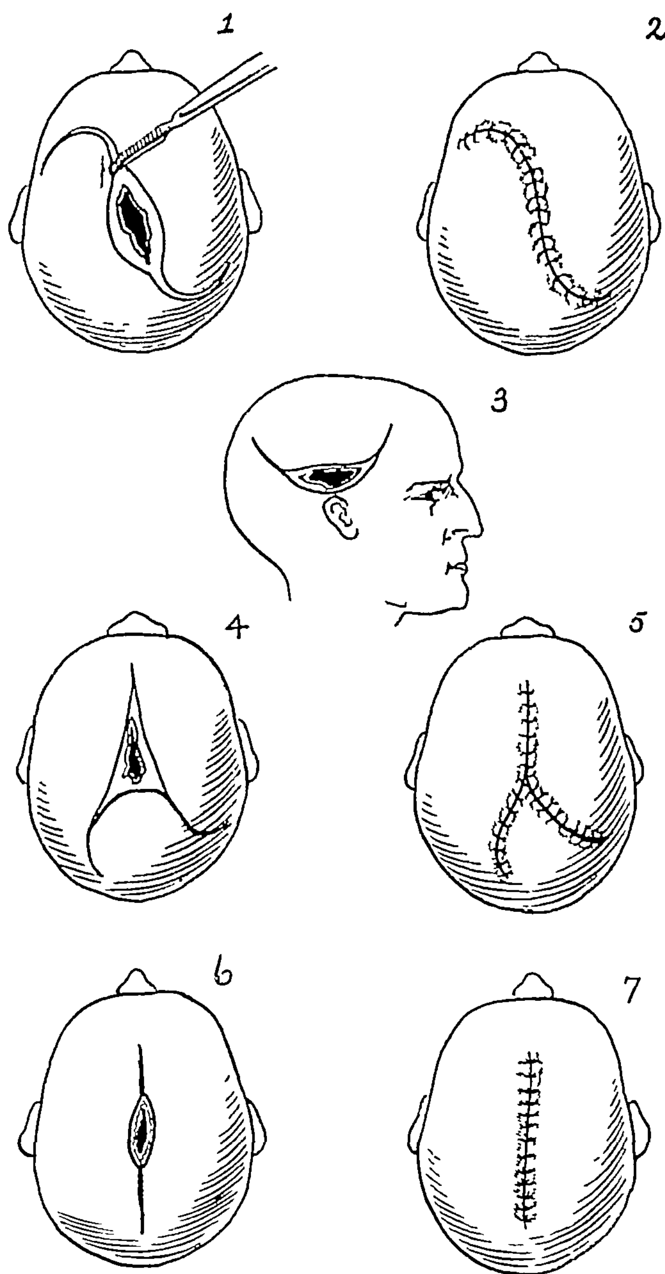


Fig 81

The better known plastic incisions of the scalp

1 and 2, The S-shaped incision showing closure 3, The flap method 4 and 5, The triradiate incision of Cushing showing closure 6 and 7, The elliptical excision showing closure

brought together by fine silk sutures passed through the galea, tension will not be too great to prevent healing When it is impossible to close the skin completely in spite of plastic incisions

the exposed part of the wound should be covered with a sheet of sterile oiled silk or amniotic membrane over which is placed a piece of gauze wrung dry after being soaked in a solution of 20 per cent. carbolic acid. Over these, the usual dressings are applied and the wound allowed to granulate. Healing later may be accelerated by Thiersch skin grafts applied to the granulating surface if this is not infected.

THE SKULL

Instruments used for the excision of skin are discarded as they are no longer sterile and the operation field is shut off with tetra cloths clamped into position with Michel clips. As all open wounds are actually or potentially infected, it is important to know the type of organism concerned as a guide for future therapy, and so a sterile lintine swab is gently rubbed into the wound and sent for immediate bacteriological examination.

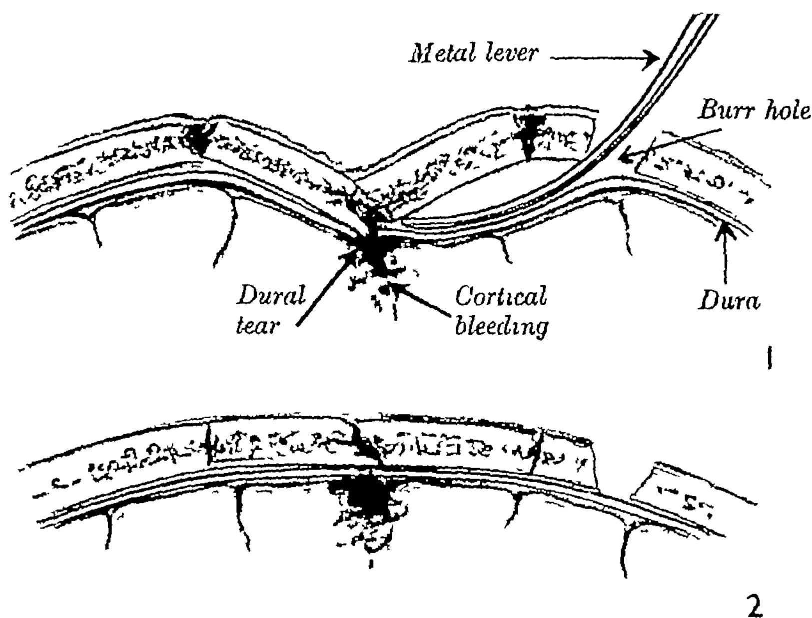


FIG 82

A method of raising an indentation

1, Shows a metal lever passed into position through a burr hole made at the edge of the depression
2, By this method a contusion of the cortex may escape recognition

Pericranium.—An encircling incision is made through the pericranium ½ in , or more if necessary, beyond the periphery of the depressed bony fragments and lacerated or presumably contaminated membrane is then swept from the bone in sheets with a sharp rongeur and removed.

Linear Fractures.—An uncontaminated linear fracture without displacement may be left undisturbed if there is no neurological

evidence of underlying injury to the brain. On the other hand, when soiling or displacement has occurred, it is safer to excise the fracture line by converting it into a narrow gutter with nibbling forceps, after a small burr hole has been sunk at one of its ends.

Indentations.—In funnel-shaped depressions, though the bone is broken the fragments are not always free. Occasionally they may swing inwards on a hinge of intact pericranium and so may be levered into position and left *in situ* if the wound has not been soiled Elevation in these cases is most easily accomplished by sinking a burr hole through sound bone at the periphery of the indentation into which a curved dissector can be introduced and used as a lever of the first order, the fulcrum being the distal edge of the burr hole (Fig. 82). When an indentation is deep or its apex spiculated as shown by radiography, it is wiser, after the bone has been raised, to cut a small gutter from the operative burr hole to the centre of the fracture so that the dura may be examined and repaired if torn (Fig 83). This is important, since an unrepaired tear of the dura or a piece of bone left sticking into the cortex is apt to lead to epilepsy

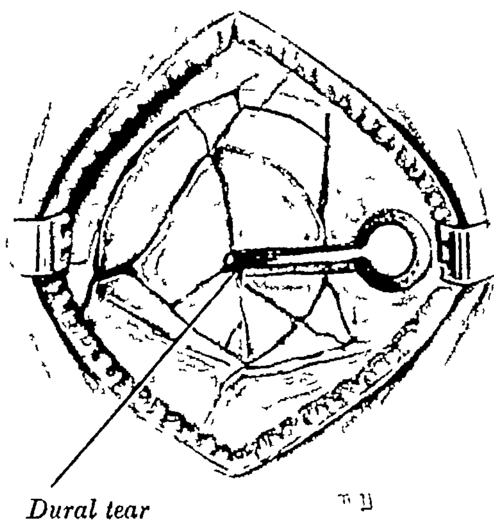


Fig 83
Exploration of the dura immediately beneath the point of an indentation

Depressed and Interlocked Fragments.—In compound, comminuted and depressed fractures of the skull it is safer to remove all loose fragments of bone, as this helps to ensure primary healing Also, loose fragments lead to prolonged suppuration should infection supervene, and have to be removed before healing will take place No qualms need be entertained on the score of free removal of bone, since repair of the skull at a later date is a relatively simple matter. Often in depressed fractures the dura is torn, and therefore when fragments of bone are interlocked great care must be taken not to lacerate the dura further with surgical instruments as an effort is made to disentangle the broken bone. When a depression is of small dimensions it is often useful to define intact dura from a burr hole sunk through sound bone at the periphery of the wound before attempting to pick up the bony fragments This allows the fracture to be approached

from without inwards and the plane of the dura always to be kept in sight In extensive depressions interlocked fragments are best removed by block resection. This is done by burring four holes through the skull at convenient points around the fracture and by lifting out broken fragments in a block after the burr holes have been connected with linear cuts made either with a de Vilbiss forceps or with a Gigli saw (Fig. 84)

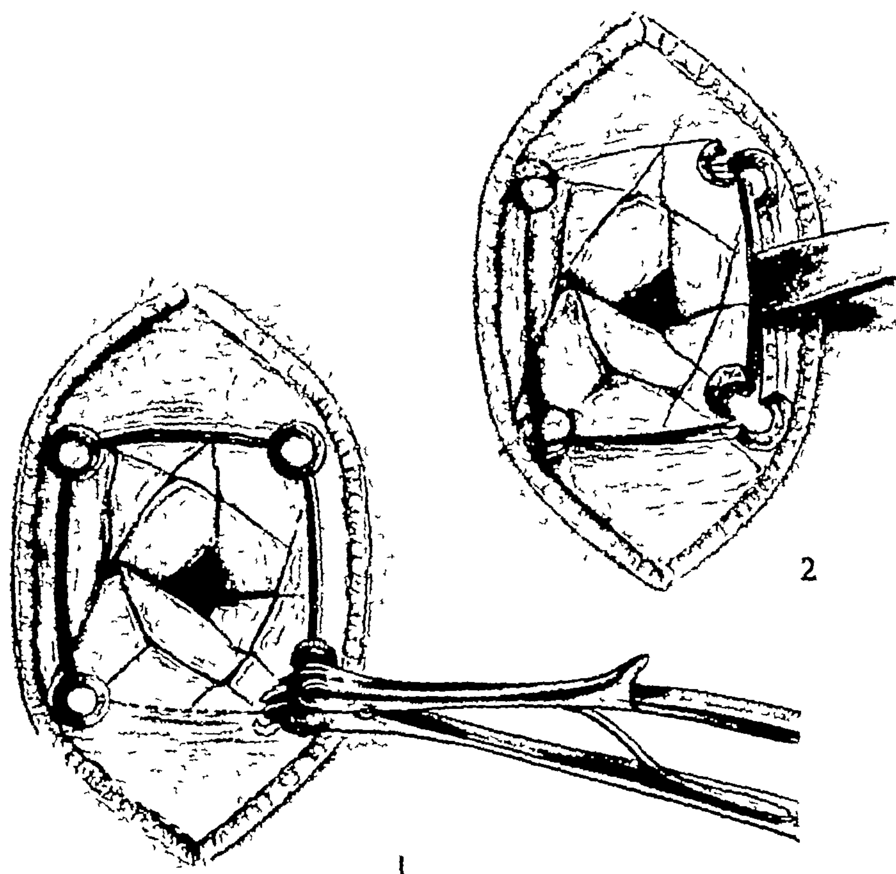


FIG 84

Removal of depressed bony fragments by means of block resection The instrument used in this case for the linear bone cut is a de Vilbiss forceps

There are, of course, exceptions to radical removal of bony fragments, and each case will have to be judged on its merits. For example, when a whole bone has been loosened but not badly displaced, it may be left in position, since the risk of its becoming infected is counterbalanced by an extensive and unsightly post-operative defect.

Cerebrospinal Rhinorrhœa.[1]—A discharge of cerebrospinal fluid from the nostrils means that a fistula has developed between

[1] Cairns, H "Injuries of the Frontal and Ethmoidal Sinuses, with Special Reference to Cerebrospinal Rhinorrhœa and Aerocele" *Jour. Laryngol E Otol*, 1937, **52**, 589

the subarachnoid space and the cavity of a paranasal air sinus or the nose. This condition is known as cerebrospinal rhinorrhœa and may present itself immediately after an injury or may not develop until some time later

Immediate Cerebrospinal Rhinorrhœa.—In severe basal injuries a discharge of cerebrospinal fluid from the nose is by no means uncommon and would be far more frequently recognised if it were not masked by concomitant bleeding. Also, patients receiving the type of injury which causes this complication often die of cerebral injury before infection has time to develop. On those occasions when patients do not succumb from laceration of the brain, meningitis may develop at any time after the injury, and no patient is free from this danger until extravasated blood has been absorbed and the fistula closed. Such healing may take many months

1. Fractures of the Ethmoid Bone.—Theoretically, a compound tear of the basal meninges owing to fracture of the ethmoid bone should be explored and repaired at once if serious infection is to be prevented. Few surgeons, however, choose to operate immediately in these cases (1) because many of the patients are severely concussed and (2) because it is believed that many cerebrospinal fluid fistulæ heal spontaneously without meningitis supervening. Moreover, it is known that the bony injury is usually extensive and lacerations of the dura ragged, so that repair, apart from the magnitude of the approach, is an operation of considerable proportions and not the simple obliteration of a single and isolated fistula.

On the other hand, Cone [1] believes that many lives are to be saved by immediate operation on all compound tears of the basal dura. He argues that shock may be treated while a patient is on the operating table, and that it is illogical to leave repair to the chances of fortune when it can be done deliberately under direct vision.

The absolute indications for immediate operative repair are —

(i) Pre-existing infection in the affected air sinus
(ii) The onset of sinusitis, osteomyelitis or meningitis.
(iii) Radiographic evidence of extensive displacement of fracture surfaces

As far as I can see the problem, operation is necessary in those cases when recovery from cerebral injury seems certain, but doubtful when the degree of concussion is such that the life of the patient is in the balance.

Once a decision to operate has been made, the best mode of

[1] Cone, W V Personal communication

approach has then to be considered Usually radiographic studies of the fracture will determine from which side of the skull a fistula should be exposed, and stereoscopic views will be found particularly useful in making a decision on this point When radiography shows that the floor of the anterior fossa on both sides has been fractured and bilateral displacement has occurred, a bilateral exposure will be necessary.

The details of the approach to the anterior fossæ are as follows ·—

(*a*) *Unilateral Approach.*—The scalp is incised along a line which starts at the glabella and then runs vertically in the midline of the forehead to within the hair-line and curves downwards and outwards into the temporal fossa to end just above the upper border of the zygoma 1 in. in front of the external auditory meatus. The skin flap thus marked out is reflected downwards and outwards. Burr holes are then sunk through the bone at points shown in the accompanying diagram and united by saw cuts (Fig 85), care being taken not to open the frontal air sinus. The danger in opening this, of course, is the introduction of unnecessary infection On the other hand, a bone flap must be as low as possible, otherwise access to the base of the skull may be so difficult that a posteriorly placed dural tear cannot be exposed Loose fragments of bone should be removed freely and the openings into the ethmoid air cells covered with a sheet of tissue taken from the fascia lata of the thigh. Dural tears are best repaired by suturing, but when this is impossible a fascial or amniotic graft should be sewn across the dural defect.

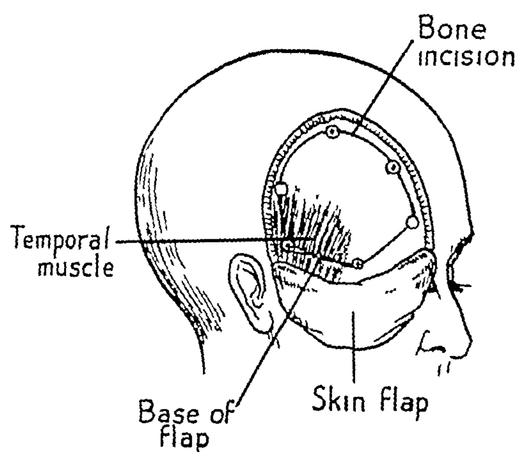


FIG 85

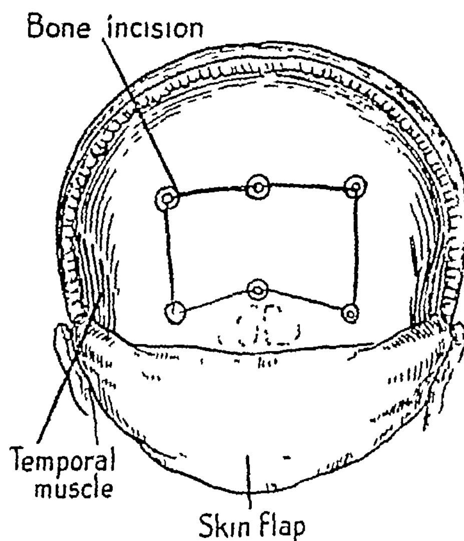
The unilateral approach to the floor of the anterior fossa

Drainage of the wound is not advisable if it can be left reasonably dry At the end of the operation the bone flap is accurately replaced and anchored in position by stitching its periosteal covering to that of the surrounding skull Finally, the skin is sewn together by two layers of sutures

(*b*) *Bilateral Approach.*—The accompanying diagram will illustrate the line of the skin incision and the area of bone to be resected (Fig. 86).

One of the difficulties of this operation is that as the bone is lifted, veins entering the superior surface of the sagittal sinus are torn and give rise to troublesome bleeding if muscle grafts have

not been previously prepared to control it. As in the above operation, loose bony fragments are removed, fascial grafts are placed across the fractured ethmoid bone and the dura repaired according to circumstances either by suturing or by grafts. The absence of a muscle or fascial attachment to the resected bone fragment makes no difference to its ability to consolidate



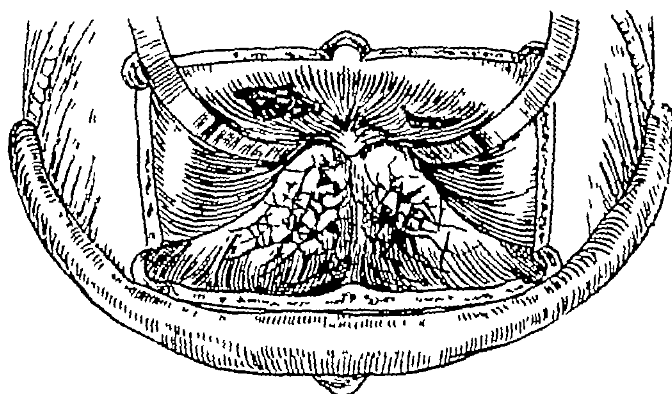


Fig 86

Exposure of a bilateral fracture of the ethmoid bone through a centrally placed bone flap

with the rest of the skull when replaced in position. Fixation of the bone may be done by periosteal suture or by sutures passed through drill holes made at corresponding and suitable sites in the skull and bone flap Again the skin is sutured together in layers.

2. Fractures involving the Frontal Air Sinus (Fig 87) — The frontal air sinus is, for obvious reasons, particularly vulnerable to injury. Even though the skin of the forehead is not broken its mucous membrane is often torn and bleeding occurs into its cavity. The danger of this happening is that the clot may become infected and lead to sinusitis or to more serious complications

such as extradural abscess. Fractures of the frontal air sinus are particularly difficult surgical problems when the overlying skin is broken, because adequate débridement of a wound necessitates complete removal of the many ramifications of the lining mucous membrane if the development of an unsightly external fistula



FIG 87

A fracture of the frontal air sinus is often best demonstrated by a routine "sinus shoot"

is to be prevented. Fractures of the posterior wall often lead to fatal complications, and the reason for this is that the dura is apt to be torn and infection and air allowed access to the meningeal spaces.

As in fractures of the ethmoid bone, decisions regarding the necessity and extent of treatment are largely guided by detailed radiographic studies.

It is unwise to lateralise the fistula of a cerebrospinal rhinorrhœa

by the nostril from which fluid is escaping, as will be seen from the description of the following case.

The schoolboy son of a doctor, while riding on his pedal cycle to work in the fields, crashed head-on into a telegraph pole and was concussed. He very quickly recovered consciousness, and no residual signs of local damage to his brain were discovered. A few days later it was noticed that cerebrospinal fluid was leaking from his nose. At this stage he was transferred to my care. Cerebrospinal fluid was found to be trickling from the left nostril but none from the right. Radiography, however, showed that a fracture of the frontal bone, which extended into the base, was confined to the right side. Also, the frontal air sinus was opaque. This opacity, presumably, was due to bleeding, and as the extravasated blood absorbed and the fundibular canal reopened, cerebrospinal fluid began to escape from the right nostril, thus declaring the true side of the fistula. Leakage from the left nostril was due to cerebrospinal fluid crossing the middle line from the right frontal air sinus to the left.

Frontal sinus injuries are best considered in two groups: (1) closed, *i.e.*, when the skin of the forehead is intact; and (2) compound, *i.e.*, when the overlying scalp is broken.[1] [2]

1. *When the Overlying Scalp is Intact*.—When the skin overlying a damaged sinus is intact, immediate operation need not be undertaken if there is no evidence that the dura has been torn and if the sinus was not diseased pre-operatively. Occasionally a surgical emphysema of the face and forehead develops, but as this usually resolves spontaneously and rarely becomes infected, incisions for its relief are unnecessary, and in fact dangerous. If infection should occur, immediate operation is necessary, and this should be done with the co-operation of an experienced rhinologist. On those occasions when radiography shows extreme displacement of the posterior wall (Fig. 88), or when a cerebrospinal rhinorrhœa has developed, exposure of the fracture and repair of the dura is necessary through a unilateral bone flap placed just above the damaged sinus.

2. *When the Overlying Skin has been Broken*.—When the scalp is broken and a clean linear fracture of the anterior wall of the frontal sinus has been found with no displacement, it is necessary only to excise the skin and remove damaged periosteum. The fracture line itself may be safely left undisturbed. When bony fragments from the anterior wall have been driven into the sinus, they must all be carefully picked out and free drainage

[1] Coleman, C. C. "Fracture of the skull involving the Paranasal Sinus and Mastoids." *J. A. M. A.*, 1937, **109**, 1613.
[2] Luckett, W. H. "Air in the Ventricles of the Brain following Fracture of the Skull." *Surg. Gyn. & Obst.*, 1917, **24**, 362.

obtained by enlarging the infundibular opening into the nose. External drainage should be avoided at all costs, as this will certainly lead to the formation of a persistent fistula Though



FIG 88

Displacement of the posterior wall of the frontal sinus of this degree is an indication for surgical exploration

great care must be taken to remove all loose bony fragments, every effort must be made to conserve the supra-orbital ridge, otherwise unsightly deformity will result. In those cases where obvious contamination of the cavity of the sinus has occurred, prolonged suppuration will result unless the lining mucosa is

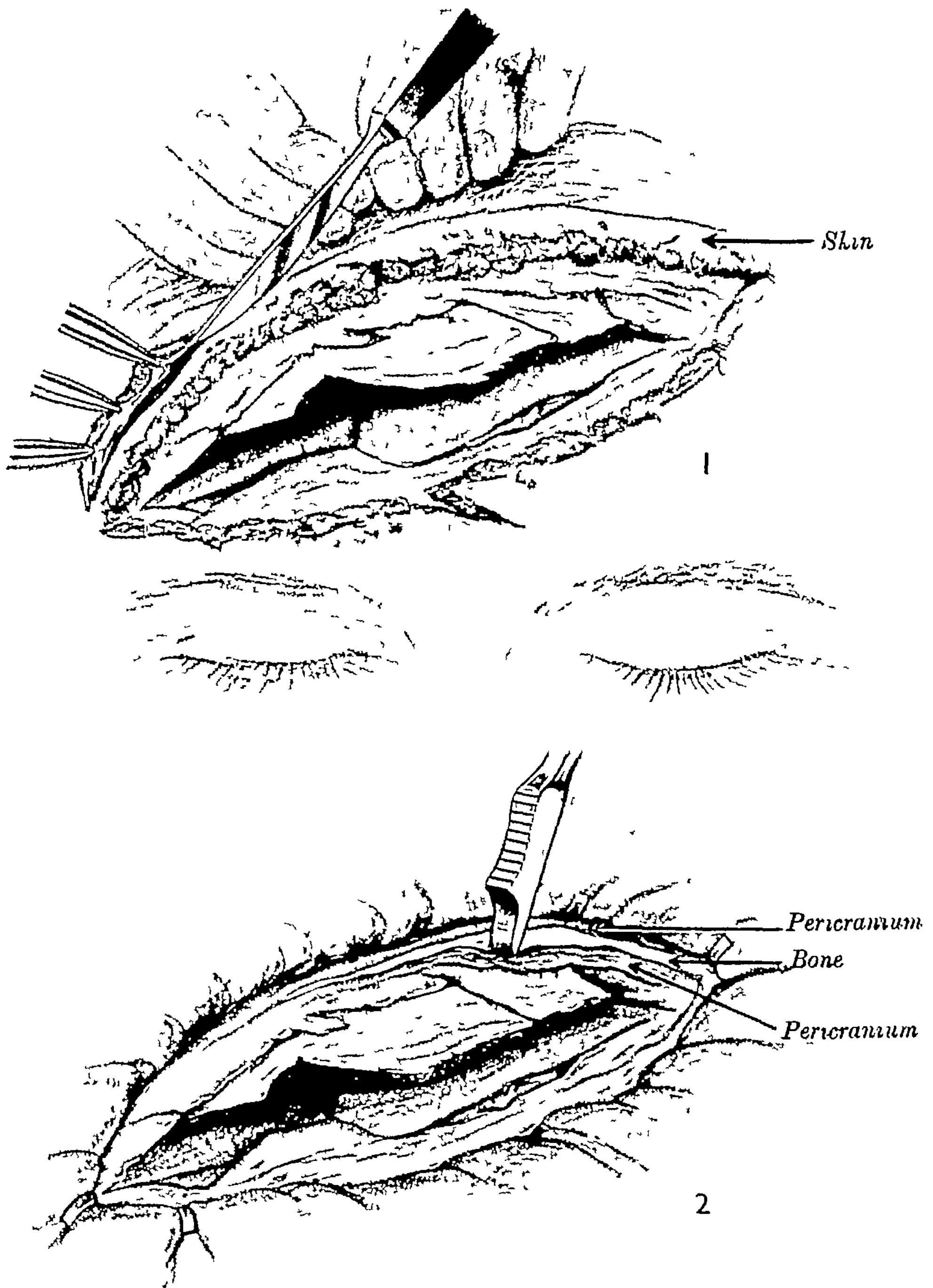


FIG 89

Excision of compound fracture of the frontal air sinus in which the dura has been lacerated

1, Excision of the skin 2, Separation of contaminated pericranium

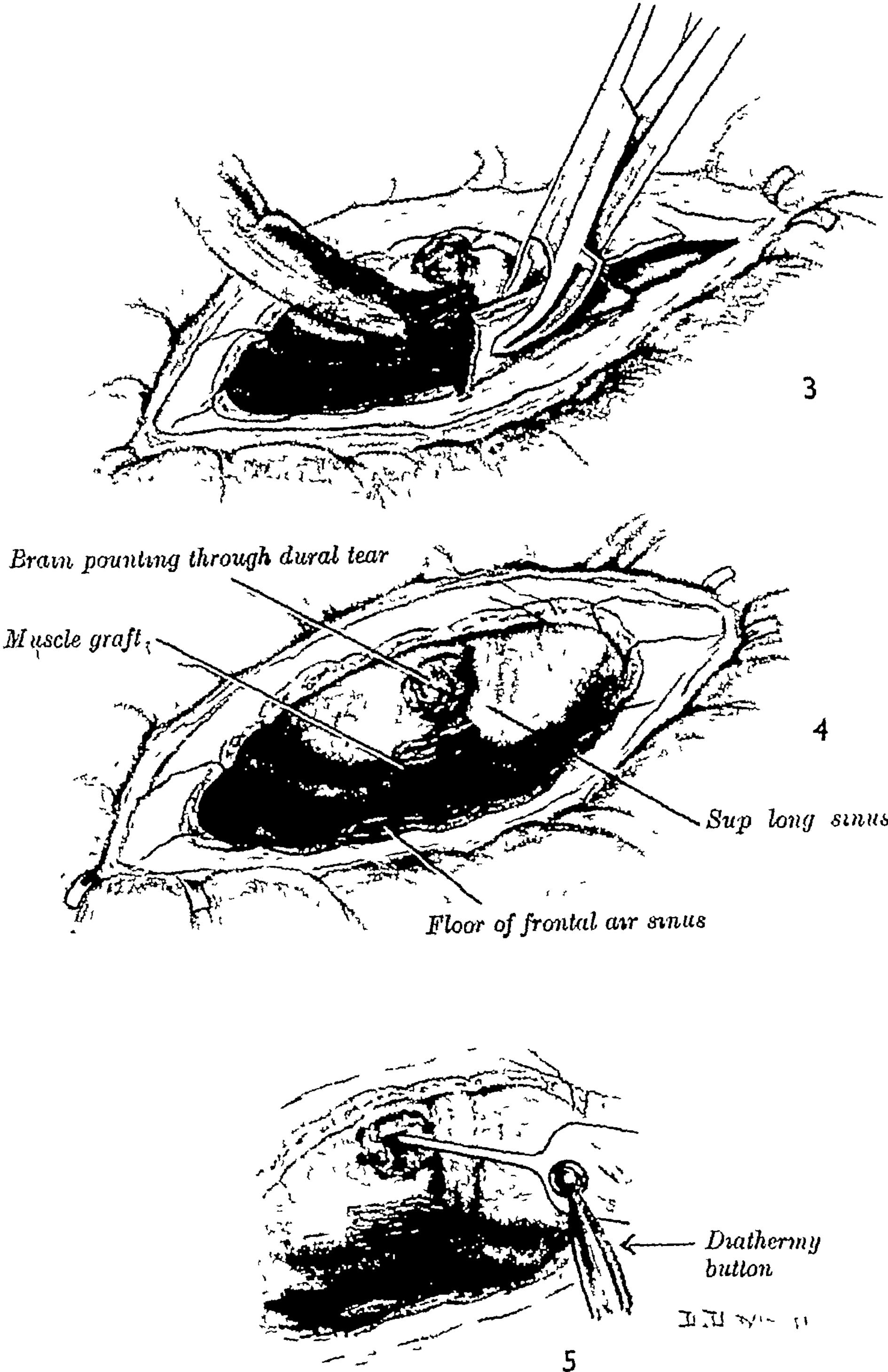


FIG 90

Excision of compound fracture of the frontal air sinus in which the dura has been lacerated

3, Removal of loose bony fragments of the posterior wall of the sinus
4, Control of bleeding from the sagittal sinus by means of a muscle graft
5, Diathermy coagulation of a ruptured cortical vessel

completely removed; this part of the operation being carried out by a combination of suction and dissection under good illumination. Without good lighting facilities and suction, operations on any sinus are apt to be imperfect.

In compound fractures of the posterior wall of the frontal sinus, whether there is a discharge of cerebrospinal fluid into the wound or not, the dura should always be explored, so that tears may be repaired immediately if this proves necessary (Figs. 89 and 90) In such cases the superficial part of the wound is excised and the mucous membrane removed before the posterior wall is disturbed so as to make the operation as aseptic as possible. Dural repairs are carried out as described previously. At the end of the operation a rubber tube passed from the operative field through the infundibulum into the nose is allowed to drain externally and is left in position for forty-eight hours.

Delayed Cerebrospinal Rhinorrhœa—Leakage of cerebrospinal fluid from the nose may occur (1) a few days after injury, (2) during the period of convalescence or (3) at any time up to a period of months or even years.

In the first two cases the fistula is probably produced at the moment of injury, but remains blocked for a time by blood clot, by a mucous plug or by an air bubble. Then, owing to some simple change in the mechanical conditions, such as alteration in the position of the head, a rise in intracranial pressure or absorption of blood, the fistula opens and cerebrospinal fluid escapes. Why leakages should occur after months or years is much more difficult to understand Some, possibly, are due to widening, as a result of the pull of cicatrisation of healing processes, of a pre-existing but exceedingly narrow fistula which has passed unnoticed. Most probably they are to be accounted for by an aseptic necrosis affecting a local area of the cribriform plate or roof of the frontal air sinus. This process of osteitis dissecans may be the result of ischæmia of the bone, consequent on rupture of its nutrient vessels as the dura (endosteum) is stripped at the time of the accident. If for any reason the dura and mucous membrane covering the affected area of bone on each of its sides become involved in the processes of necrosis, a fistula will result

Diagnosis is simple. A colourless fluid trickles from the nose in drops and this discharge may be made more profuse by straining or by bending the head forward. Occasionally a leakage of cerebrospinal fluid may be confused with a watery discharge, either from the lachrymal gland or mucous membrane of the nose Since cerebrospinal fluid is the only fluid which contains sugar, a chemical analysis in these cases is all that is necessary to distinguish cerebrospinal fluid from secretions arising within the nose.

The side of the nose from which the discharge comes may lateralise the fistula and intranasal inspection will show whether it is situated anteriorly or posteriorly. When the fluid is seen to be trickling from above the middle turbinate, the fistula passes through the ethmoid or sphenoidal air cells, but when the fluid

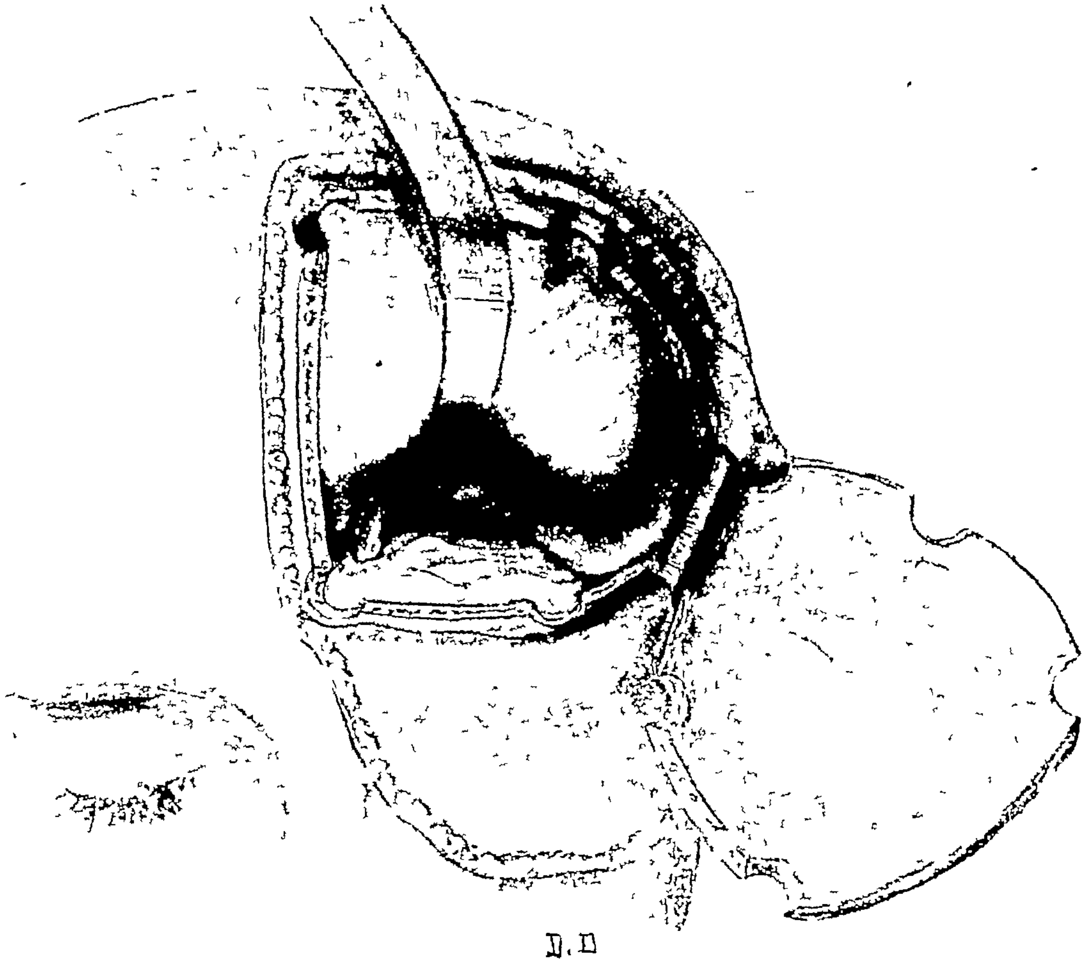


Fig 91

Exposure of the fistula in a case of delayed cerebrospinal rhinorrhœa through a unilateral transfrontal flap

drips from beneath the anterior end of the middle turbinate the fistula is more anteriorly placed and passes through the frontal sinus.

If necessary, methylene blue injected into the ventricles will definitely determine the presence of a fistula, since in this case the discharge from the nose will be coloured blue.

Treatment.—Treatment consists of repair of the fistula through a unilateral frontal approach as described previously (Fig. 91)

Recently Julian Taylor [1] has shown that an intradural approach to a chronic fistula has advantages over the better-known extradural approach; by this means manipulations in a possible septic field are avoided and a view across the middle line is easily obtained if this proves necessary.

Craniomastoid Injuries.—As noted in a previous chapter, bleeding from the ear does not necessarily mean that the dura mater has been torn; in fact it does not necessarily indicate a fracture, since the blood may be coming purely from a ruptured ear-drum. The only unequivocal sign that the cerebral membranes have been torn is discharge of cerebrospinal fluid or brain tissue from the external auditory meatus.

Whether a compound fracture of the petrous bone is present or not, and the drum is broken, great care must be taken to avoid infection. To ensure this, coagulated blood should be removed at once from the external auditory meatus by swabbing, but never by syringing. Then a small wisp of carbolic gauze should be placed in the external auditory canal and a sterile dressing bandaged over the ear. Syringing and tight packing of the external auditory meatus are unwise because of the danger of washing infection inwards or of causing blood to collect intracranially. When cerebrospinal fluid is present in the discharge, prophylactic chemotherapy must be started to prevent meningitis Healing of a cerebrospinal fluid fistula may be promoted by limiting fluid intake and by daily lumbar punctures.

Further details of the anatomy and complications of mastoid injuries will be given later.

THE DURA MATER

The treatment of open wounds confined to the scalp and skull is a relatively simple matter. On the other hand, when the dura and brain have been damaged, strict observance of neurosurgical principles becomes necessary if good results are to be obtained. Not only has the immediate problem of life and death to be considered, but possible sequels such as epilepsy and brain abscess must be minimised by correct procedure. In cases where there is a rise in intracranial pressure, an injudicious opening of the dura mater, for example, or an imperfectly planned operation on the brain may lead to extensive cerebral damage.

When the Dura Mater has not been Torn.—Intact dura should never be opened when the superficial parts of a wound are obviously infected, otherwise meningitis or encephalitis will result. It is

[1] Taylor, J Paper read by Mr Eden at the meeting of the Neurosurgical Society, Oxford, July 1941

also dangerous to open the dura when a wound is heavily contaminated. Contamination implies the presence of foreign material in a wound; infection, that inflammation due to bacterial action has already started.

When a patient is unconscious and a clot can be seen through the dura, it should be evacuated if this can be done aseptically. A small incision, not more than ½ in. in length, is made through the dura, the nozzle of a sucker inserted and the blood removed. The dura is then carefully sutured.

When a patient is unconscious and intradural tension is increased, as judged by finger-tip palpation, and a subdural clot cannot be seen, cerebrospinal fluid should be withdrawn via the lumbar theca until the pressure is reduced to normal. In those cases when intracranial pressure remains high in spite of lumbar drainage a decompression is necessary, and this, according to circumstances, may have to be made on the side of the skull opposite to the wound.

In a conscious patient, whether a subdural clot can be seen or not, it is not absolutely necessary to open the dura, and if there is any doubt about infection it is better not to do so.

When the Dura Mater has been Torn.—On those occasions when it is known that the dura is torn, a lumbar-puncture needle with its stylet in position should be introduced into the lumbar theca before the start of the operation. This necessitates the patient lying on his side, but there is no disadvantage in this position, since his head can easily be manipulated into a position which will make any wound accessible By withdrawal of the stylet of the puncture needle and spinal drainage, intracranial tension can be lowered at any stage of the operation if found necessary. In this way intradural manipulations may be facilitated and harmful cerebral herniations prevented.

The first stage of an operation is to expose the whole of the dural tear by removing the bone around it, either by piecemeal nibbling or by block resection. When fully exposed, a dural tear should be sutured if elastic retraction of the membrane or loss of tissue has not made this impossible (Figs. 92 and 93). A ragged tear may be sparingly excised if diathermy is available to seal off the meningeal spaces afterwards, and such sealing is effected by drawing a diathermy button along the excised dural edge. On no account must dura be extensively excised, as this may reopen meningeal spaces which have already been sealed off by natural processes, or allow a cerebral hernia to form.

When dura has been lost, the defect may be repaired by a fascial or amniotic graft if it is considered that the wound will heal by first intention. However, if there is the slightest danger

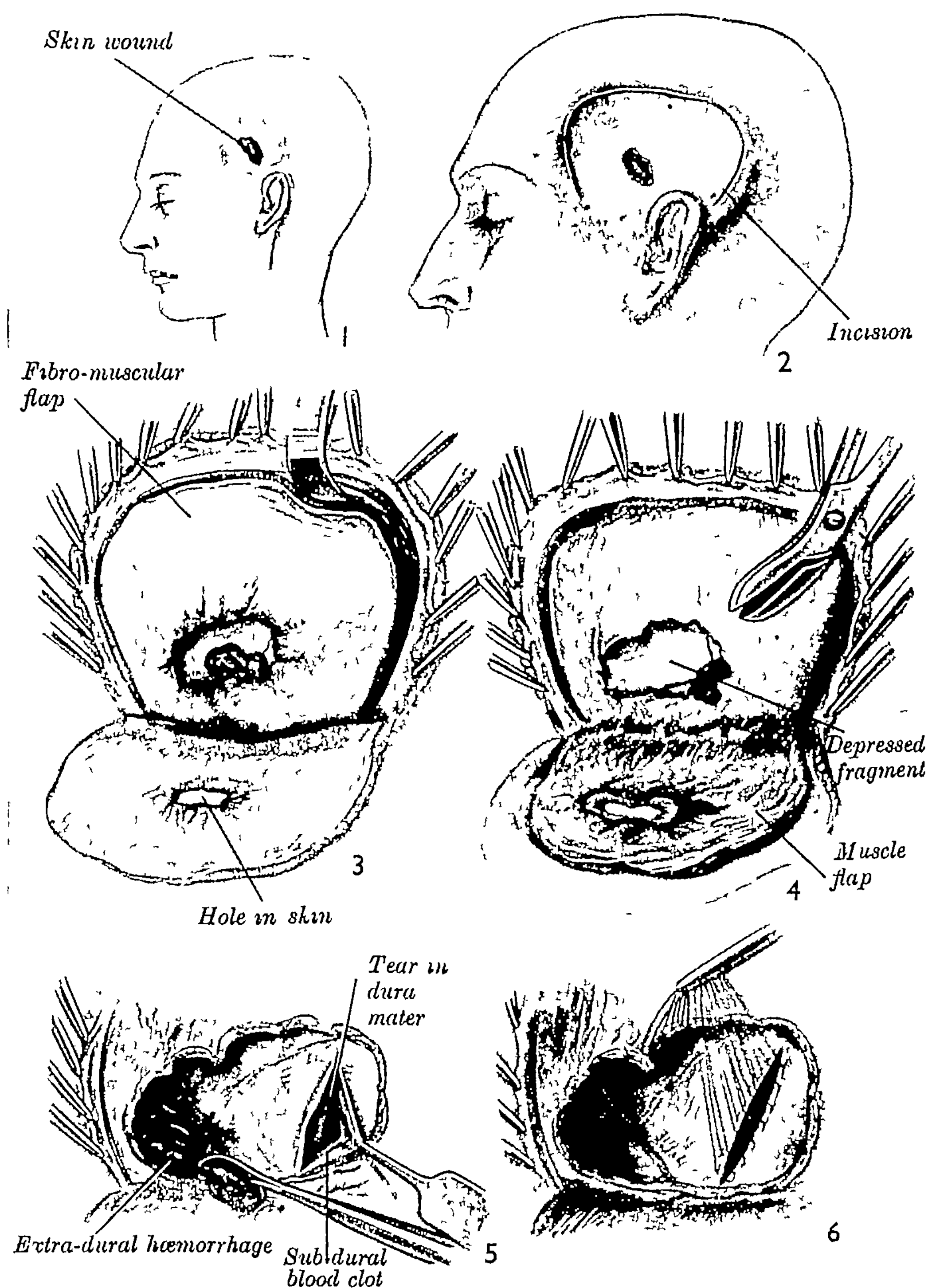


FIG 92

A method of excising a compound dural tear in the temporal region

1, The site of the wound 2, A semicircular skin flap was used for the exposure
3, The skin flap turned down exposing a tear in the temporal muscle
4, Reflection of the fibromuscular flap subperiosteally
5, Exposure of dural tear and removal of an extradural clot A ruptured meningeal vessel was sealed by diathermy coagulation 6, Repair of dural tear by sutures

of infection the skin only should be pulled together and dural repair left for secondary operation.

Recently Penfield[1] has shown that amniotic membrane causes less gliosis when in contact with injured brain than pericranium or fascia lata. This, therefore, is the tissue of choice in the repair

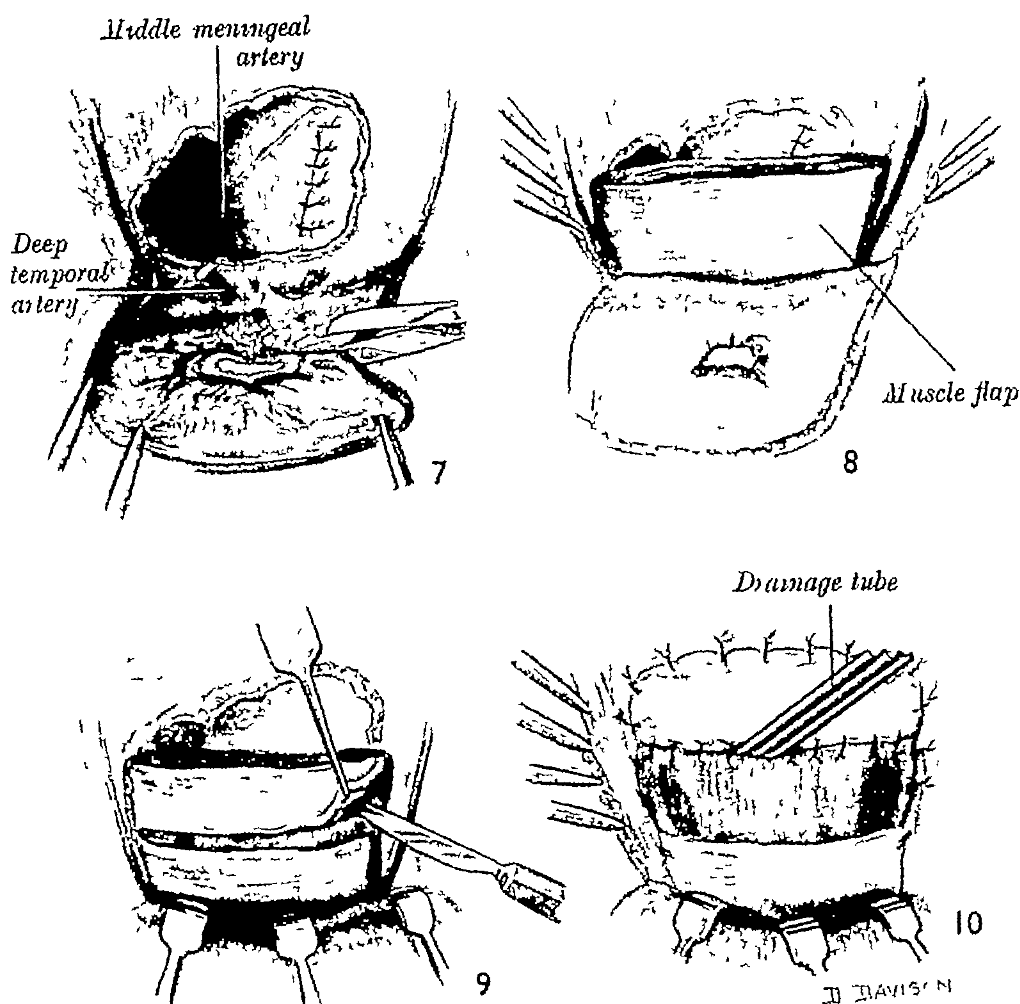


FIG 93

A method of excising a compound dural tear in the temporal region

7, Removal of injured part of muscle
8, The shortened temporal muscle left the upper part of the bone defect uncovered
9 and 10, Reformation of the fibromuscular flap by plastic repair. Finally, the skin wound was excised and its skin flap sewn into position with two layers of sutures

of dural deficits. Its preparation is simple. Sheets of amnion are obtained at a healthy birth, sterilised by boiling and stored in alcohol. Tendency to crumple may be overcome by preparing it between flat glass plates. At the time of the operation it is cut to the required shape, lain across the defect and tensed beneath

[1] Chao, Y C, Humphreys, S S, and Penfield, W "New Method of preventing Adhesions, use of Amnioplastin after Craniotomy" *Brit Med Jour*, 1940, 1, 517

the dural edges. In my experience attempts to suture it in position have not always been successful because it is so friable.

When amniotic membrane is not available and a large graft is required, fascia lata will have to be used. In this case a linear incision is made on the outer side of the thigh and a rectangle or square of fascia removed according to the size of the dural defect. This is later cut to the required shape and sewn to the dural edges with its smooth surface facing the brain. Small dural defects may be repaired by pericranium taken from a healthy part of the wound.

THE BRAIN

The special conditions of the brain demand a special operative technique, the details of which must be observed meticulously if a successful operation is to be performed. Manipulations must be carried out with the utmost gentleness and great care taken not to injure healthy brain tissue adjacent to the operative field by heavy retraction as a wound track is opened for inspection. Any kind of rough handling of the brain will lead to swelling, which may not only impede surgical procedure but also may prove fatal. All bleeding vessels must be sealed either by coagulation, silver clips or by muscle grafts, for until this has been done it is useless to close a wound, since a post-operative clot is certain to collect and compress the brain. When a vessel is torn it is unwise to press a finger or a swab heavily on to it to stop bleeding, as consequent displacement of the brain will lead to rupture of distant and inaccessible veins entering the dural sinuses. The bleeding vessel should be isolated by suction and sealed by what seems the most suitable method.

Treatment of a cerebral wound is designed to remove dead tissue, extravasated blood, fragments of bone and foreign bodies, and these objects must be kept clearly in mind and nothing further attempted if unnecessary damage to healthy parts of the brain is to be avoided. In superficial wounds, foreign bodies may be picked out with dissecting forceps and loose tissue washed away with jets of warm saline projected from a C Ryle syringe. When a deep laceration occurs, the wound track should be opened and exposed in its whole length by means of flat metal brain retractors (Fig 94). To do this successfully, skilled assistance and efficient lighting are necessary. Wet lintine swabs placed along the walls of the wound track will protect the brain tissue from damage by the metal retractors Débridement is then carried out by suction. A glass tube of 3-mm. lumen diameter is used as the sucker nozzle. This is passed along the opened wound track and weak suction used, so that healthy brain tissue is not lifted

at the same time as damaged matter is removed. Dead brain tissue, extravasated blood and small bony fragments will pass along the lumen of the tube, and these should be caught in a suitable trap, otherwise they will block the sucker. Medium-sized foreign bodies will be drawn into the opening of the glass sucker and may be removed when the sucker is withdrawn. Large bony fragments or missiles may be identified and loosened by suction and then removed with forceps under direct vision. Healthy

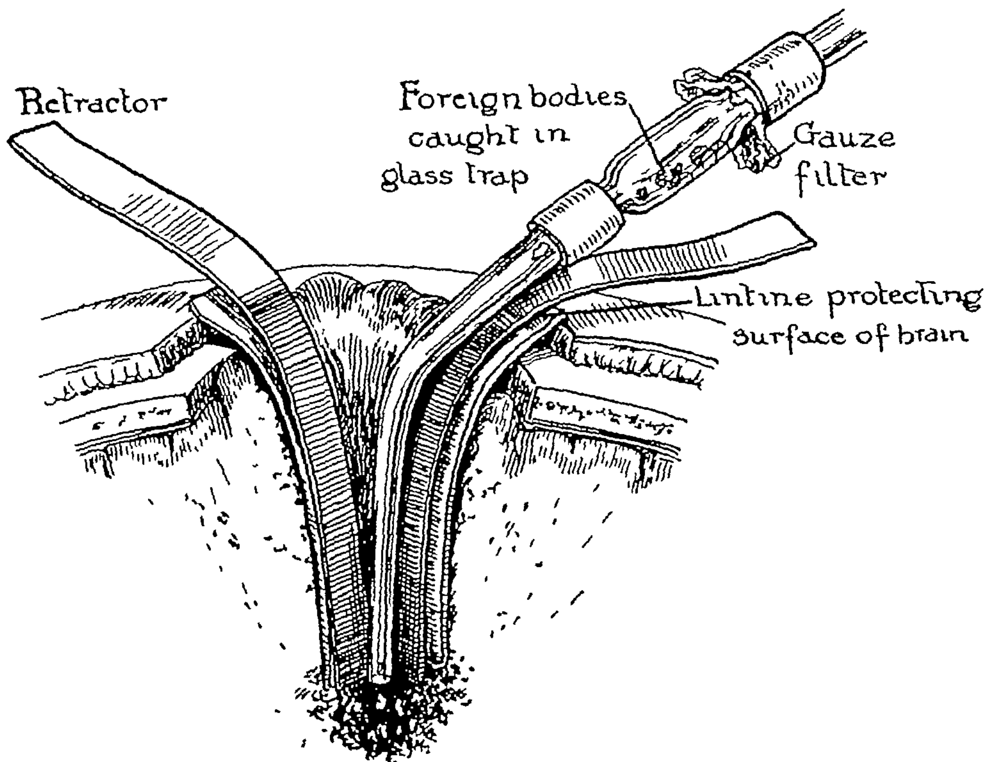


Fig 94
Débridement of a wound track

tissue must never be removed in the hope of getting a clean block resection, as this is likely to lead to spread of infection or to unnecessary neural deficits. In particular, débridement in the region of the motor cortex must be done conservatively and with great care if hemiplegia is not to be the result.

RETAINED MISSILES

A bullet or bomb fragment is often deeply embedded in the brain, and whether it should or should not be removed immediately depends on its accessibility. When it can be reached safely along a wound track without infliction of further damage to the brain it should be removed, but on no occasion should a separate entrance through the skull be made for its extraction. During débridement a bullet may be encountered in the track of the wound, and in

these cases it is extracted with forceps. For extraction of metal fragments from the bottom of a wound which are difficult to grasp with forceps an electric magnet is useful. The steel bar of the magnet is passed into the wound, the current switched on and after a few seconds the magnet withdrawn. A few volts only are needed to activate the magnet, otherwise pieces of metal may be dragged through healthy tissue from a distance, with resulting laceration of the brain tissue. It must be remembered that a magnet is not a powerful instrument to be applied to the surface of the brain but is purely an adjunct to débridement and never replaces it. When a metal missile is not easily accessible, it should be left *in situ*, because it rarely gives rise to a brain abscess and its presence does not materially influence the incidence of epilepsy. An epileptogenic focus tends to lie in the superficial or cortical part of a wound track and not in the area immediately surrounding a foreign body.

Removal of a foreign body at a later date may be necessary if complications such as infection or epilepsy develop. In these cases the operative entrance is determined by accessibility. For example, a bullet is approached from the surface nearest to which it is lying, provided that this is a silent area. It would, of course, obviously be wrong to incise the motor cortex in order to gain access to deep tissues. When infection is already present in the superficial layers of a wound, the only indication for removal of a foreign body is when it is giving rise to an expanding abscess.

DURAL VENOUS SINUSES

Repair of a torn dural venous sinus is always difficult and often hazardous. Therefore, whenever a depressed fracture is seen to lie over the superior longitudinal or lateral sinus, certain pre-operative precautions should be taken to anticipate bleeding. The patient's blood group is determined and a pint of blood obtained pre-operatively. After primary shock has been treated, the theatre is fully prepared and the patient, if not already unconscious, is given an injection of morphia. When asleep or quiet, his head and one leg are carefully shaved and made aseptic. The patient is then placed on the table and every means used to lower cerebral venous pressure. His head is raised and fixed above the level of the rest of the body. When a neurosurgical table is available the patient should be tilted into a sitting position. The blood at all stages must be fully oxygenated by keeping the tongue forwards and by oxygen bubbled into the nasopharynx through a small tube. Local anæsthesia is the method of choice. General anæsthesia by inhalation must always be avoided, as

this blows up the venous sinus and often causes serious bleeding before the surgeon is in a position to control it. When a patient is restless, intravenous pentothal is the best method of anæsthesia.

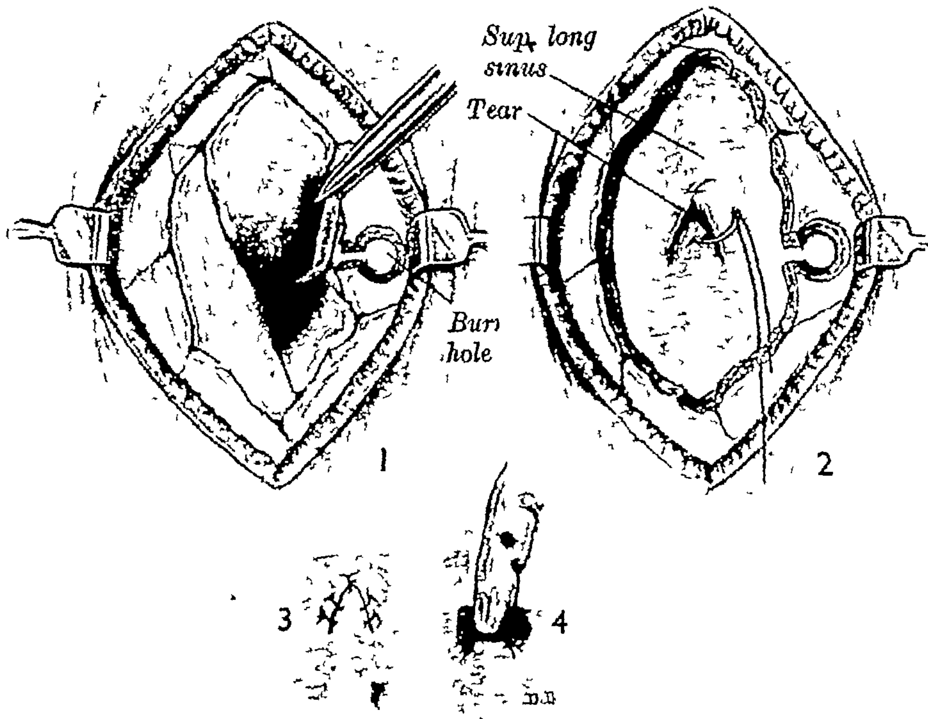


FIG 95

Exposure and repair of a tear of the wall of the superior sagittal sinus

1, Removal of the bony fragments starting from a burr hole peripherally placed
2, Suture of the tear in sinus wall 3, Completion of suturing
4, The suture lines are finally covered with a muscle graft

Before commencing the operation on the head the anterior compartment of the leg is opened and muscle grafts prepared. At the same time the internal saphenous vein is exposed and a saline drip started so that, when necessary, blood may be given without delay. Then the superficial layers of the head wound are excised Bony fragments are lifted out singly, starting at the point farthest away from the sinus until the dural tear is fully uncovered. One of the great dangers is that severe bleeding may start before all the bone necessary for exposure has been removed. When this happens it is best to proceed with the bone removal as expeditiously as possible.

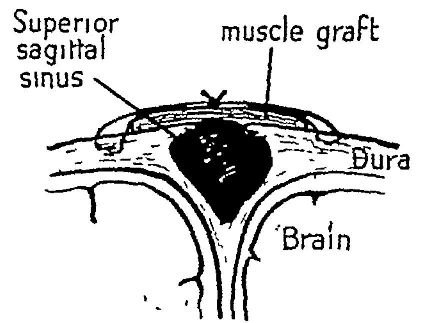


FIG 96

A method of holding a muscle graft in position over a dural sinus by means of suturing

On those occasions when a sinus is found to be completely severed, each end must be clamped with forceps and firmly ligated

with stout silk thread. When a tear is extensive, but not complete, the edges of the wound should be drawn together with a continuous or interrupted suture and the suture line covered with a muscle graft (Fig. 95). When a tear is small it can often be controlled by muscle grafts alone, but these may have to be sewn into position as shown in Fig. 96.

DRAINAGE AND DRESSINGS

A small wound may be closed without drainage after all bleeding points have been sealed and when it is thought to be free from infection. Drainage, on the other hand, is the safer procedure when a wound is extensive or has been obviously heavily contaminated. When it is decided that drainage is necessary, a

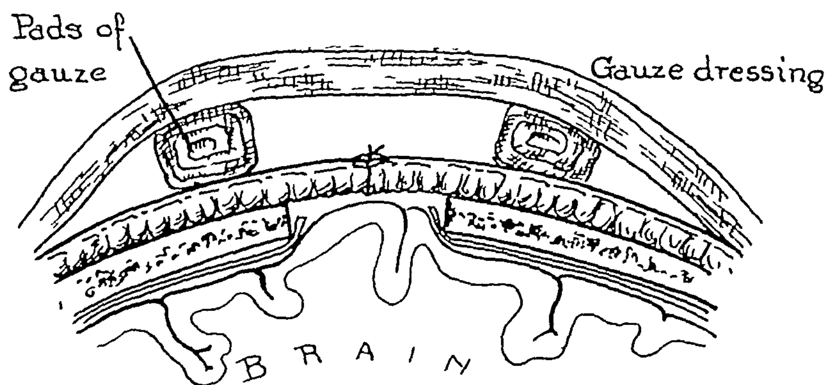


FIG 97
When bone has been lost a built-up dressing will avoid dangerous pressure on the brain

narrow strip of corrugated rubber or fine tubing is placed beneath the scalp and brought out through what will be the most dependent part of the wound, as judged by the position in which the patient will subsequently be nursed. Intradural drainage must never be employed. Also, drainage tubes should not be allowed to issue from those positions where healing is apt to be delayed. Before applying sterile dressings, blood should be carefully and completely washed from the scalp to avoid infection. The wound is then dressed with layers of gauze soaked in surgical spirit. In those cases where the dura has been left widely open the brain must be protected from pressure by building up the dressings at the periphery of the wound over sound bone (Fig. 97).

When a tube has been inserted to drain possible post-operative oozings of blood it should be removed within thirty-six hours. It should, however. be left in as long as necessary when infection is expected. In the absence of infection the opening left by a drainage tube ought to be closed with a suture to promote rapid

healing and to avoid the formation of a fistula. When a wound suppurates free drainage must be provided at once, if necessary through suitable stab incisions, since pus under tension will lead to spreading encephalitis. In those cases when the dura mater is open and cerebrospinal fluid or a brain hernia is stretching a wound, repeated lumbar punctures will relieve the tension and allow the skin to heal firmly.

CEREBRAL FUNGUS

In those cases when the dura mater is torn and the intracranial pressure is high, the cortex of the brain will bulge through the opening as a hernia When suppuration has prevented primary healing and the skin wound has broken down, the surface of the brain becomes exposed and a cerebral fungus ensues (Fig. 98). This is a troublesome complication and requires careful treatment. The fungus should be covered with a sterile sheet of amnion or gutta-percha tissue and pressure avoided by building up gauze dressings at the periphery over sound bone. Any effort to amputate the herniated brain is not merely useless but dangerous. So long as infection is present, further brain tissue will continue to ooze out, however much of it is removed, and after a time the ventricle will enter the hernia, which if opened will result in ventriculitis; also, unnecessary amounts of brain tissue are destroyed if amputations are attempted. The infection is treated by chemotherapy. Intracranial tension is minimised by repeated lumbar punctures and free withdrawal of the cerebrospinal fluid until the cerebral œdema has subsided. A natural repair may result by epithelium growing in from the skin edges to form a covering Often, however, this covering is incomplete and a plastic operation may be necessary later to cover the hernia.



FIG 98
A large cerebral fungus due to faulty surgery

MENINGITIS AND ENCEPHALITIS

Brain tissue possesses considerable bactericidal powers and is able to overcome mild infections or to localise suppurative processes by the formation of barriers of neuroglial and fibrous tissue.

Thus penetrating wounds of the brain do not necessarily lead to a meningitis or to a spreading encephalitis. In particular, spread of infection over the cortex is soon limited by the obliteration of the subarachnoid spaces either by swelling of the brain or by meningeal adhesions in the region of a wound. Unfortunately the natural barriers against infection cease at the ventricular walls, and once organisms gain access to the ventricular cavities they are freely washed into the cerebrospinal fluid spaces and diffuse meningitis results (Fig. 99).

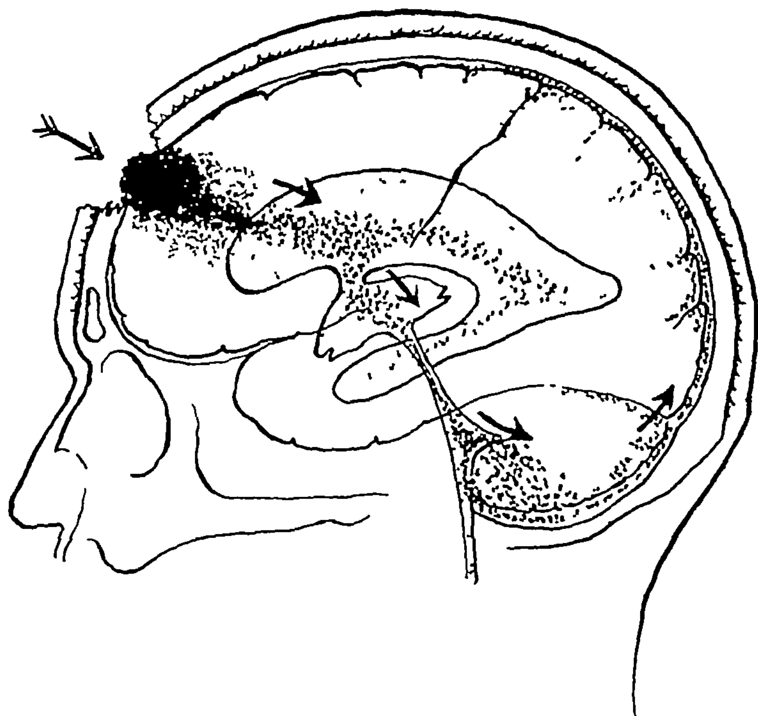


FIG. 99

Ventriculitis is the common cause of a fatal termination resulting from an infected wound of the brain (*After Jefferson*)

When an infective intracranial complication develops, lumbar puncture should be done at once and a specimen of cerebrospinal fluid sent for bacteriological and cytological examination. Suitable and extensive chemotherapy is started as soon as a diagnosis of meningitis or cerebritis is made, and this is supplemented later by suitable serum injections when the causative organism has been isolated Also, cerebrospinal fluid should be freely withdrawn twice daily by lumbar punctures. After the second puncture this is usually a tedious procedure, since fluid is apt to drain away very slowly owing to increase of its viscosity consequent on the presence of products of inflammation. Often it takes half an hour to collect more than 30 c.c., but it is time well spent, since drainage relieves pain and encourages the circulation of the cerebrospinal

fluid. Cisternal puncture may be used as an alternative route for drainage when the lumbar theca becomes blocked with adhesions, as it so often does. Toxæmia is treated by the administration of copious amounts of fluids; when a patient is vomiting and unable to drink freely, a cannula should be tied into a vein and glucose saline given as a continuous drip.

It is important not to give excessive amounts of fluids intravenously, otherwise transudations will occur into the pleural or peritoneal cavities with fatal results. For this reason not more than 5 pints of fluid should be given in one day. Improvement or retrogression may be judged in cases of post-traumatic meningitis not only on clinical signs but also on the information given by cell counts done at the time of each lumbar puncture. Even in fulminating cases of infection many lives can be saved by persistent and energetic treatment, and no case should be given up as lost until every known type of therapy has been tried.

THE VALUE OF LOCAL ANTISEPTICS AND CHEMOTHERAPY

During the last war numerous types and combinations of local antiseptics were used in the treatment of open wounds in an attempt to prevent or overcome infection B.I P.P., solutions of hypochlorinates and mercurial salts were the chemical substances most commonly employed, and they are still in use to-day. The final consensus of surgical opinion, based on 1914-18 war-time experience, was that early and careful débridement of a wound was the essential factor in attaining primary healing, and that the local application of antiseptics to a wound was of minimal importance. Antiseptics are certainly of no value when infection has once developed, as they cannot penetrate surface exudations and coverings to destroy the bacteria which, by this time, are already embedded in the deeper tissues As Dorothy Russell has pointed out, Jefferson and Monro omitted to mention the use of local application of antiseptics when they discussed treatment of compound wounds of the head, and this, of course, is an indirect commentary on their opinion of their efficacy Certainly their views were largely based on peace-time experiences, but in my opinion the same principles apply to wounds received in air raids, the essential for primary healing being thorough excision of a wound—which implies the necessity of adequate neurosurgical facilities—before infection has become established.[1] The ideal antiseptic must have antibacterial properties when placed in contact with the tissues of the body and should be innocuous to tissues locally and not dangerously toxic if absorbed

[1] Neurosurgical facilities do not imply facilities only for a neurosurgeon

CHAPTER VI

THE RESULTS OF INJURY TO SPECIAL PARTS OF THE BRAIN AND SKULL

THE VISUAL PATHWAYS

THE **Eyes.**—Rupture of the globe of the eye, as a result of cranial injury of the closed type, is almost unknown. In my clinical series of 1,000 cases it did not occur on a single occasion, which is a striking commentary on nature's skill in protecting important organs. No doubt this immunity of the eye from serious injury under peace-time conditions is due to (1) the protection it receives from the thick bony rim of the orbit, (2) the relative smallness of the eye which allows it to move away from injury whenever the orbital cavity is deformed, (3) the mode of accident, flat rather than pointed objects being responsible for the violence.

In road and industrial accidents, however, hæmorrhages into the globe of the eye are by no means uncommon, and bleeding may occur (1) into the anterior chamber, (2) into the vitreous, (3) into the retina or (4) between the coats of the eye.[1] Subhyaloid hæmorrhages may also occur, but these are usually associated with subarachnoid bleeding and are thought to be caused by blood being forced along the optic sheath from the intracranial cavity.

In air-raid casualties penetrating wounds of the eye unfortunately are common, and are due either to bomb fragments or to small bodies such as spicules of glass being blown forcibly through the air. Displacement of the axis of the eye occurs when the orbit is deformed by fractures involving the face or forehead. As it is the orbital floor rather than the roof which breaks, the eye is usually displaced downwards Outward and downward displacement are often associated (Fig 100), but upward and inward displacements are exceedingly rare. Diplopia, however, is by no means an invariable sequel of displacements of the eye as is the case when ocular neuro-mechanisms are impaired

[1] Special number on "War Wounds of Eye and Orbit" *Post-graduate Medical Journal*, 1940, 16

Since treatment of penetrating wounds of the eye is the concern of an ophthalmologist, nothing further need be said on this subject here save that treatment is urgent and no time should be lost before skilled assistance is sought

The disability of temporary diplopia may be avoided by covering the affected eye with a shade, but as this interferes with binocular vision such occupations as the driving of cars or the working of a machine in a factory should be suspended. Persistent diplopia, due to displacement, which resists orthoptic treatment, may necessitate reconstruction of the orbit and replacement of the eye before the visual images can be satisfactorily fused. Such operations, of course, are best done by plastic surgeons.



Fig 100

Downward and outward displacement of the eye consequent on fracture of the orbit This man suffered from persistent diplopia

The Optic Nerves.—Gunshot wounds of the orbit may bruise or sever one or both optic nerves, producing blindness or defects in the visual field of a pattern that can readily be explained by the circumstances of penetration

In closed injuries, on the other hand, the mechanism of optic nerve damage is not so obvious, and more will have to be said on this subject In my series of acute head injuries the incidence of post-traumatic blindness was about 0 5 per cent, and in 500 cases examined for litigation purposes it was found on only seven occasions In Russell's[1] series of 600 cases, evidence of injury to the optic nerve occurred eight times.

It is rather surprising that damage to the optic nerve is so infrequent, particularly as cranial injuries are so commonly basal in position As Rawlings[2] has shown, the infrequency of optic nerve damage is possibly due to the fact that frontal fractures which converge on the pituitary fossa cross the sphenoidal fissure rather than the optic canal. This explanation, however, is not entirely satisfactory, because in Vance's[3] series of 512 necropsies,

[1] Russell, W R "Injury to Cranial Nerves including the Optic Nerves and Chiasma" 'Brock's Injuries of the Skull, Brain and Spinal Cord," chap v. Baillière, Tindall & Cox London, 1940

[2] Rawlings, L B "Surgery of the Skull and Brain" London, 1912

[3] Vance, B M "Fractures of the Skull" *Arch Surg*, 1927, **14**, 1023

the optic canal was fractured in 10 per cent. of cases and in von Hoelder's series of eighty-eight basal fractures, in 61 per cent A fractured optic canal, of course, does not necessarily mean that the optic nerve has been contused, since it is displacement of fractured surfaces that causes injury to soft parts, and this does not occur in every case. Moreover, detachments of the anterior clinoid processes are usually associated with fatal degrees of violence It may be presumed, therefore, that optic nerve injury would be more common if cases of the severer types of injury survived

The actual cause of blindness is a subject of great controversy. Lindsay Rea [1] inclines to the view that it is due to blood within the optic nerve sheath, and although he did not actually make the statement he presumably means by compression. At autopsy, intravaginal bleeding is certainly a common finding, and the extravasated blood may come from a subarachnoid hæmorrhage or from rupture of the retinal arteries and veins which cross to the nerve from its sheath. Against this view is the non-occurrence of optic nerve blindness in subarachnoid hæmorrhages caused by spontaneous rupture of a congenital aneurysm of the circle of Willis. When blindness does occur in these cases it is due to a subhyaloid hæmorrhage which can be seen on ophthalmoscopy and not to compression of the nerve fibres. Traquair believes that rupture of the small vessels supplying the nerve explains its loss of function This may explain some but by no means all cases of blindness. Stretching of the nerve fibres is another possible mode of damage, but this mechanism must be rare, otherwise avulsion of the nerve would occur more frequently than it does.

Cone [2] has shown by histological methods that blindness may be caused by contusion of the nerve within the optic canal, and as will be shown later, this is in my experience an important mechanism of injury.

Clinical findings are variable and depend on degree and extent of injury and on the presence or absence of lesions in neighbouring structures, such as the oculomotor and sympathetic nerves The eye may be completely blind and the direct light reflex absent In spite of this, the pupil is not necessarily dilated, since it may be kept contracted by the consensual reflex. An appreciation of light or of hand movements may be all that is left of visual acuity, in these cases the direct reflex is present though sluggish. Incomplete injuries produce all types of visual field defects For example, central vision may be spared or absent, and peripheral loss may be quadrantic or scotomatous

Tabulated below are the main findings in seven cases of blindness

[1] Lindsay Rea, R "Neuro-ophthalmology" Heinemann Ltd London, 1938

[2] Cone, W V. Personal communication

following head injury. In six cases the blindness was unilateral and in one bilateral, suggesting injury of the optic chiasma, but the presence of fractures in both orbits was against this view. A fracture of the optic canal was demonstrable by radiography in three only of the seven cases, although fracture of the anterior fossa or orbit was present in six. In six cases the optic disc was pale, with clean-cut edges typical of primary atrophy, and in one only was the disc normal in colour. No doubt the absence of atrophy in this latter case can be explained by the fact that the disc was seen only three days after injury; three weeks later this disc also showed signs of atrophy. In the six unilateral cases the blood vessels of the disc were found to be normal on comparison with those of the opposite side, meaning that the arterial supply of the retina was unimpaired. The veins, moreover, were not engorged nor the disc œdematous, as would be expected if the venous return of the retina had been obstructed by an intravaginal hæmorrhage. In one case that came to autopsy the nerve fibres had been compressed in the optic canal and there was an unmistakable indentation at the point where this had occurred (Table).

TABLE

Age	Sex	Nature of Injury	Severity of Injury	Side	Visual Acuity	Size of Pupil	Direct Light Reflex	Consensual Light Reflex	Discs.	Vessels of Discs	Prognosis.	Fracture of Frontal Bone.	Fracture of Optic Foramen
21	M	Motor car accident	Unconscious few hours	L.	Shadows only	Medium and equal	Sluggish	+	Atrophic	Normal	Bad	+	-
53	M	Fall in epileptic fit	Not concussed	L.	"	"		+	Normal then atrophic	"		-	+
35	M	Hit by handle of crane	Not unconscious	L.	"	"	"	+	Atrophic	"		+	-
56	M	Blow on head	"	R.	Totally blind	"	Absent	+	"	"	"	+	+
43	M.	Motor car accident	Unconscious twelve hours	L.	Shadows only	"	Sluggish	+	"	"	"	+	-
13	F	Blow on head	Unconscious several hours	R	"	Fully dilated	Absent	-			"	+	+
28	M	Fall on head	Unconscious few hours	L. & R.	Blind Vision present in inferior nasal quadrant only	Fully dilated Medium dilated	Eccentric contraction		"	Abnormally small on both sides		+	-

To give an opinion on prognosis of optic nerve blindness in the early stages is difficult. Perimetry may be helpful. If a field defect shades at its periphery, the sight in the shading field may recover because traumatic œdema and not irremediable structural injury may be the cause. Theoretically, treatment of optic nerve blindness should be immediate exposure of the nerve at the clinoid process and decompression of the optic canal. In practice, of course, this is never done because an accurate diagnosis cannot be made as long as the patient is unconscious, and when blindness is discovered it is usually too late for treatment to be of any avail. Moreover, there is no guarantee that early operation

will accomplish anything useful, the injury being in the nature of a contusion rather than of a compression.

"Delayed post-traumatic blindness" has occasionally been described. It may result from strangling of the nerve by fibrous



FIG 101 —The right optic canal, as shown by radiography

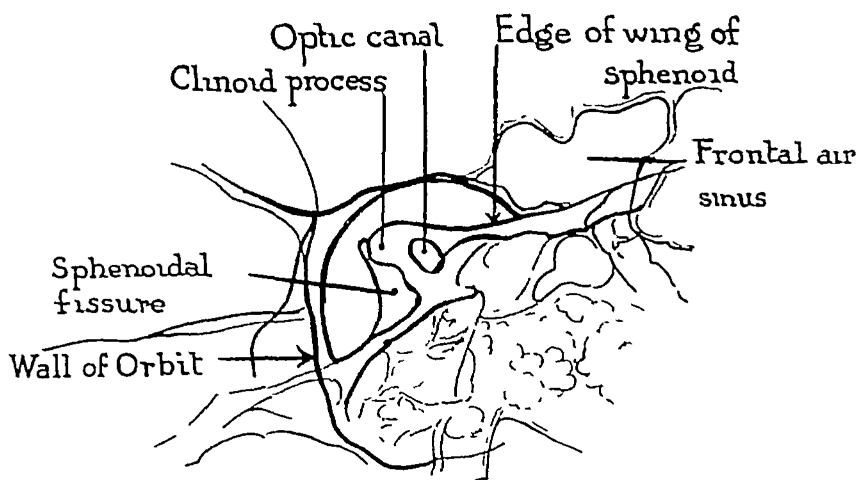


FIG 102 —Key to Fig 101

tissue or callus as a fractured optic canal heals or from a progressive arachnoiditis Operative treatment is always indicated in these cases and consists of removal of the roof of the optic canal after the floor of the anterior fossa has been exposed through an osteoplastic flap (Figs 101-104)



FIG 103

Narrowing of the left optic canal due to fracture The optic nerve in this case had been contused and the eye was blind

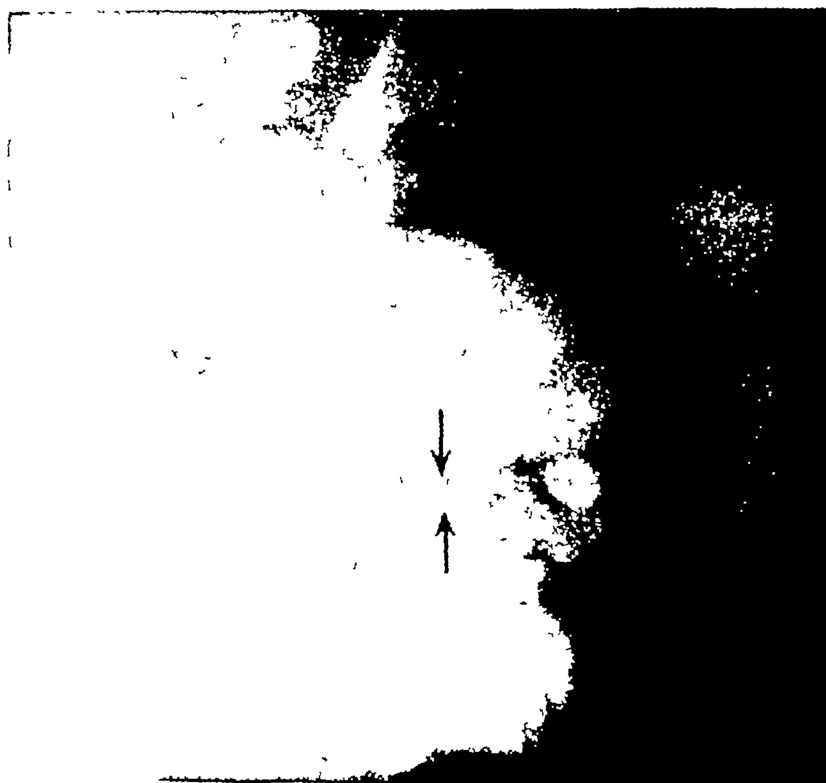


FIG 104

Fracture of the base of the left anterior clinoid process The eye was blind on this side

The Intracranial Pathways (Fig. 105)—It is surprising how rarely the intracranial pathways are damaged in view of their

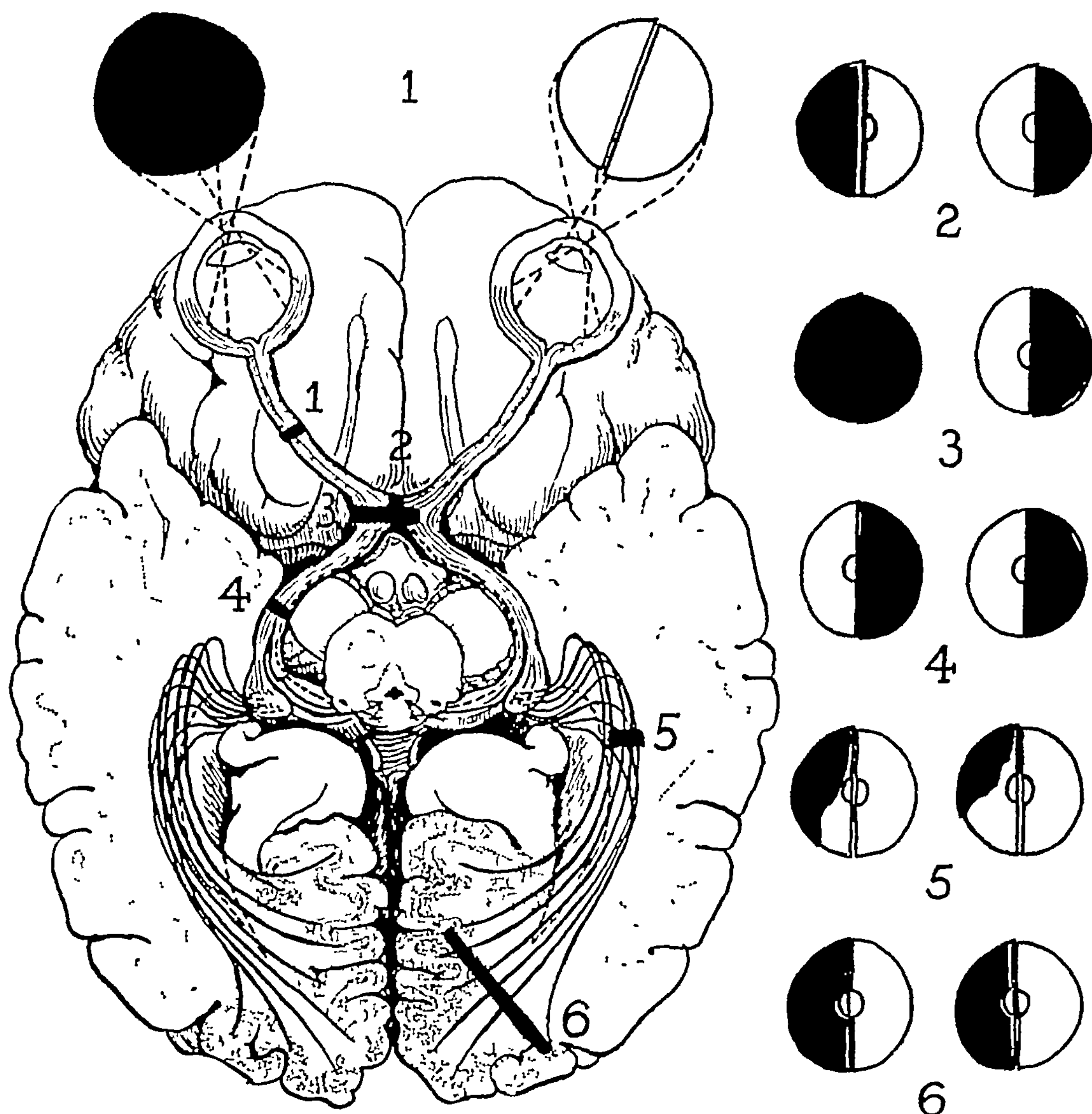


FIG 105

Injuries of the visual pathways

1, Injury to the optic nerve produces unilateral blindness
2 and 3, Injuries of the optic chiasma may produce bitemporal hemianopia or complete blindness in one eye and blindness in the other temporal field
4, Injury of the optic tract produces homonymous hemianopia of the congruous type with loss of half macular vision
5, Injury to the optic radiations produces homonymous hemianopia of the incongruous type
6, Injury to the poles of the occipital lobes produces homonymous hemianopia of the congruous type, usually with sparing of macular vision, since macular vision is widely represented in the occipital lobe or is anteriorly placed

extent and of the exposed position of the optic tracts as they lie at the base of the brain

The optic chiasma is sometimes affected, but rarely by contusion Loss of vision in these cases, as Traquair, Dott and

Russell[1] have shown, is due to rupture of the small chiasmal vessels with resulting ischæmia of the nerve fibres The most characteristic defect is a bilateral loss in the temporal fields of vision (bitemporal hemianopia) or a total blindness in one eye and a temporal loss in the other.

Damage to an optic tract is a very rare injury indeed and shows as a congruous homonymous hemianopia

Lacerations of the occipital lobes or of the optic radiations, with resulting loss of vision in the contralateral visual fields, are sometimes caused by compound depressed fractures or perforating wounds When the occipital lobe is concerned (Fig. 106), the

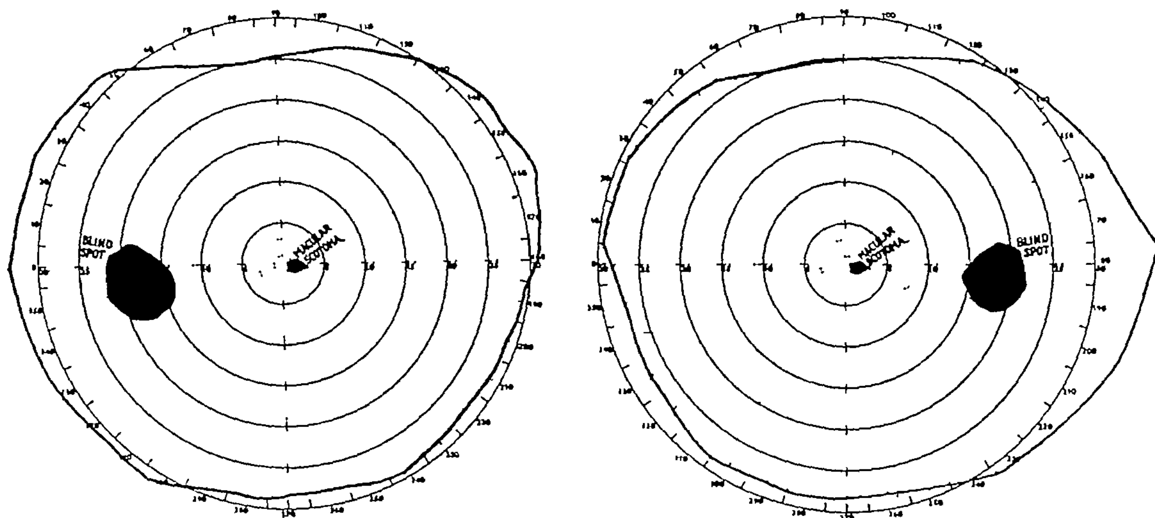


Fig 106

Name Dr M *Perimetrist* Dr J S Arkle *Test Object* 2/1000 white

A homonymous scotoma involving central or macular vision resulting from a fall on the back of the head This is a very unusual finding, since macular vision is thought to have a very wide representation in the visual cortex or to lie in the region of the calcarine fissure itself and not at the tip of the occipital lobe

pattern of the visual loss is symmetrical in both fields (congruous homonymous hemianopia), whereas in injuries of the optic radiations the loss in each field is asymmetrical (incongruous homonymous hemianopia)

INJURIES OF THE CRANIAL NERVES

Sense of Smell.—Loss of smell follows injury of the head in about 5 per cent of cases, and is particularly liable to occur when an injuring force has been applied to the head anteriorly or through the bones of the upper part of the face In itself it is of no great importance except in those people whose occupations are dependent to some extent on their sense of smell

It is, however, a serious disability when the power of

[1] Traquair, H M, Dott, N M, and Russell, W R "Traumatic Lesions of the Optic Chiasma" *Brain*, 1935, **58**, 398

appreciating aromas of food or wines is lost, as this interferes with one of the primary pleasures of life One of the peculiar findings in head injuries is that although loss of smell may apparently be complete, that of taste (other than of salt, sour, bitter, sweet) may not be impaired. Possibly the ambiguous findings in these cases may be explained by the fact that sense of smell is not completely absent but is so faint that a gaseous stimulus passing through the anterior nares cannot be appreciated, whereas it may be possible when it arises in the nasopharynx In many cases of loss of smell it is possible to demonstrate a fracture of the anterior fossa by radiography. In those cases when fracture or dislocation of the cribriform plate is not present the olfactory filaments have probably been torn as the brain moved across the base of the skull at the time of accident. Avulsion or tearing of the olfactory tract, described and illustrated by Cairns,[1] is a very rare occurrence, and at fifty autopsies I did not find it in a single case. Occasionally the olfactory centres may be contused and lead to uncinate attacks. In one of my cases, epilepsy of the convulsive type following injury was preceded by a horrible smell the nature of which could not be described save that it was offensive and nauseating In such an uncinate attack I have seen a man spit out and shake his head in disgust at the nastiness and reality of the sensation

Prognosis in anosmia is doubtful Improvement to some extent is usual, but full recovery is rare when loss of smell has been complete On no occasion have I known of recovery when complete loss of smell has persisted for more than three months

A very useful guide in testing the veracity of a patient's statement as regards loss of sense of smell is that sensations dependent on the integrity of the trigeminal nerve should be present if this nerve has not been damaged, and such damage may readily be shown by loss of common sensation. Therefore if a patient is analysing sensations carefully and giving accurate answers, he ought to be able to appreciate pungent vapours in the nose and to recognise salt or vinegar on the tongue The significance of these tests should never be allowed to come to the knowledge of the public, otherwise they will lose much of their usefulness in helping medico-legal assessments to be made

The Oculomotor Mechanism.—Damage to the third, fourth and sixth cranial nerves in closed head injuries usually results either from fractures of the orbit or from stretching of the nerves in the posterior fossa as the brain stem is displaced at the moment of violence Penetrating wounds which damage the ocular nerves

[1] Cairns, H "Injuries of the Frontal and Ethmoidal Sinuses with Special Reference to Cerebrospinal Rhinorrhœa and Aeroceles " *Jour Laryng & Otol*, 1937, 52, 589

other than within the orbit usually cause fatal injuries by severing large intracranial vessels

A complete paralysis of any ocular muscle supplied by the third or sixth nerve usually causes an obvious squint, and diagnosis of the affected muscle may be made with certainty (Fig. 107). On the other hand, when a muscle is merely paresed it is often impossible to detect malposition or even imperfect movement of the eye concerned. In these cases a careful analysis of the resulting diplopia is necessary if the affected muscle is to be discovered Diagnosis depends on the fact that when an eye is made to look in the direction of pull of the weak muscle, true and false images become more widely separated. Such separation is most easily recognised if the eyes are covered with different coloured glasses and a white light used for the object of fixation, so that false and true images are seen in different colours. Doubt is thrown on a patient's observations when he complains of the continuance of diplopia when an object is placed in the extreme temporal field of one eye and outside the nasal field of the other Such placing of the object of fixation is, of course, possible because the projection of the nose limits the nasal field of either eye, and this limitation can be mapped out by means of direct confrontation. Tests are carried out as follows First, the patient's affected eye muscle is detected by alterations of the movements of the true and false images. Then the patient is asked to look directly into the examiner's eyes and a small rounded object such as a white hatpin is held at an equal distance from the patient's and examiner's eyes. The examiner now closes his eye which is opposite to the patient's faulty eye and keeps the other open. The patient is then asked to look at the head of the hatpin and the examiner does likewise The hatpin is then moved towards the side of the closed eye until it just disappears from the nasal field of the open eye If accurate observations are being made by the patient the complaint of diplopia should cease when the object

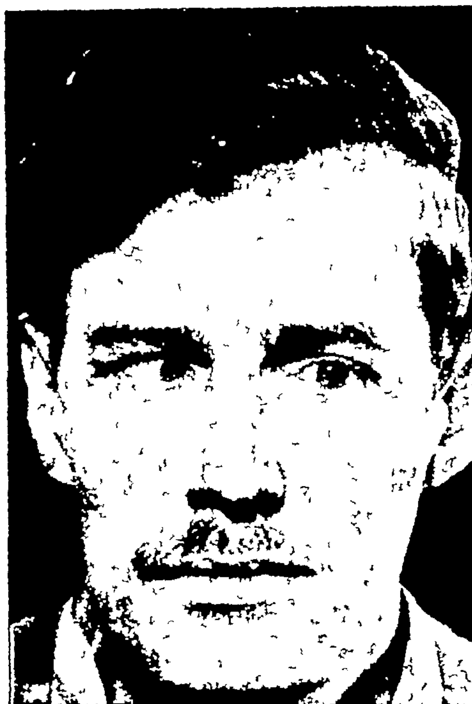


Fig 107

Paralysis of the external rectus muscle of the left eye The man is looking towards his left ear Complete recovery occurred three months after this photograph was taken

of fixation passes out of range of the examiner's nasal field, as this corresponds with the patient's nasal field of vision.

Diplopia, the result of damage of an ocular muscle or of contusions of the brain stem, usually recovers spontaneously within a few weeks or months. Persistent diplopia from any cause is exceedingly rare and necessitates treatment in an orthoptic clinic.

Injuries of the Trigeminus.—Areas of anæsthesia about the head and face often are the result of severance of branches of the trigeminus by scalp wounds received at the time of accident. Most commonly the supra-orbital and supratrochlear nerves are divided in the forehead just above the supra-orbital notch. This leads to anæsthesia of the anterior two-thirds of the scalp on the same side. Contusion or laceration of one or more of the three divisions of the trigeminus or of its large branches may occur in osseous injuries as the nerves course through channels or openings in the bone to reach the face from the intracranial cavity. For example, a fracture of the superior maxilla may lead to damage of the infra-orbital nerve with resulting anæsthesia of the cheek Fractures of the lower jaw with displacement lead to severance of the dental nerve and numbness of the lower lip, teeth and jaw. In closed head injuries the Gasserian ganglion is occasionally injured by basal fractures that cross the tip of the petrous bone Hæmorrhages into the ganglion sheath occasionally produce a condition which is indistinguishable from herpes zoster. The results of gunshot wounds are heterogeneous, and varying degrees of injury may be inflicted on the trigeminal nerve at any point from its posterior nerve root to its peripheral filaments.

Apart from incised and penetrating wounds, complete loss of sensation in any area of the face or scalp is rare Usually sensation is partially and not completely destroyed; in these cases recovery of some degree may be expected, although normal sensation rarely returns.

It must be remembered that, subjectively, numbness is a very real and unpleasant sensation, the characteristic feature of which may be intense cold, burning, swelling or a creeping feeling under the skin Occasionally an extremely painful interstitial trigeminal neuritis or paroxysmal neuralgia may be the result of an injury to the Gasserian ganglion

Objectively, as sensation returns to an anæsthetic area, the skin may become hypersensitive and remain so for the rest of the patient's life. In medico-legal circles the question is often asked whether anæsthesia of the scalp is likely to lead to loss of or change of colour of the hair In my experience of a series of 300 cases of nerve section or alcohol block done for trigeminal neuralgia this complication has never been observed

The Syndrome of the Jugular Foramen.—The glossopharyngeal, vagus and spinal accessory nerves pass through the jugular foramen to enter the neck in this order from before backwards On account of their protected position they are very rarely injured, and when this does occur the violence is usually overwhelming and leads to fatal results

Injury to the Hypoglossal Nerve.—In closed head injuries I have never seen a case of contusion or laceration of the hypoglossal nerve alone. Usually injury to the hypoglossal nerve is associated with the syndrome of the jugular fossa, and this combination has been described by Collet[1] following a penetrating wound of the posterior fossa and by Galand[2] in the case of a patient who fractured the base of the skull by diving into shallow water.

INJURIES OF THE EAR AND FACIAL NERVES

Deafness and Facial Paralysis.—As stated in Chapter I, the body of the petrous bone offers stout resistance to injuring forces and deflects to its base most fractures which run towards it either from the middle or from the posterior fossa This, no doubt, is the reason why fractures involve the middle ear far more frequently than the internal ear

Forces of great magnitude occasionally break the body of the petrous bone transversely and sever the seventh and eighth nerves as they lie within the internal auditory canal (Fig 108). Such injuries, however, are usually fatal and so are of no clinical importance

In any case, fractures involving the middle ear are those with which the surgeon is chiefly concerned, not only because they may result in deafness and facial paralysis but also because they may result in otitis media and meningitis, for which active treatment is possible Fractures involving the middle ear may be divided into two distinct groups. (1) those confined to the base and (2) those which are continuation fractures from the vault

Middle-ear deafness may result from either type and is caused by one or a combination of the following factors (1) blood in the tympanum; (2) dislocation of the ossicles, (3) blocking of the eustachian tube; (4) rupture of the membrana tympani, and (5) interference with the action of the tensor tympani The membrana tympani is not invariably ruptured when bleeding has

[1] Collet, M "Sur un Nouveau Syndrome Paralytique Pharyngo-Larynge par Blessure de guerre" (Hemiplegie glosso-laryngo-scapulo-pharyngée) *Lyon Med*, 1915, **124**, 121

[2] Galand, G 'Syndrome total des Quatre Derniers Nerfs Craniens (Collet) avec Paralysie du Sympathetique ou Syndrome de l'Espace Parotidien Posterieure (Villaret)" *Ann d'oto-laryng*, 1932, 1288

occurred into the middle ear, and in these cases auroscopic examination will show the drum to be bulging and discoloured. Diagnosis of the exact cause of deafness following head injuries is always difficult and is essentially a problem for an otologist.



Fig 108

Fracture of the body of the petrous bone, as shown in the above radiograph, is uncommon In this case there was deafness of the internal-ear type and complete facial paralysis The man died of meningitis ten days after injury

Injury to the head with or without demonstrable petromastoid damage is very serious in people who were losing their hearing before accident since any pre-existing degenerative or chronic inflammatory process affecting the ear may be considerably accelerated thereby.

Prognosis is always doubtful in middle-ear deafness and definitely bad when the internal ear is affected.[1] According to Davis,[2] if improvement of hearing does not begin within eight weeks following an accident the chances of recovery are small.

In my experience facial paralysis has been more commonly due to fractures running into the base from the vault than to those confined to the base. In three cases, two at operation and one at autopsy, I have been able to trace in detail the course of a fracture



FIG 109

This is the type of fracture which runs across the roof of the middle ear and produces facial paralysis

which led to paralysis. On each occasion the fracture started in the squama of the temporal bone or in the posterior inferior angle of the parietal bone (Fig. 109) and then ran downwards into the base of the mastoid bone just behind the posterior wall of the external auditory canal At this point it turned inwards across the roof of the middle ear and ended in the facial canal distal to the geniculate ganglion (Fig. 110). This, I believe, is the typical fracture which leads to damage of the facial nerve.

[1] "Discussion on Injuries of the Ear" *Proc Roy Soc Med*, 1940, 34.
[2] Davis, E D D *Proc Roy Soc Med*, March 1931, 69

Facial paralysis may be immediate or delayed. Immediate facial paralysis is the result of contusion or compression of the

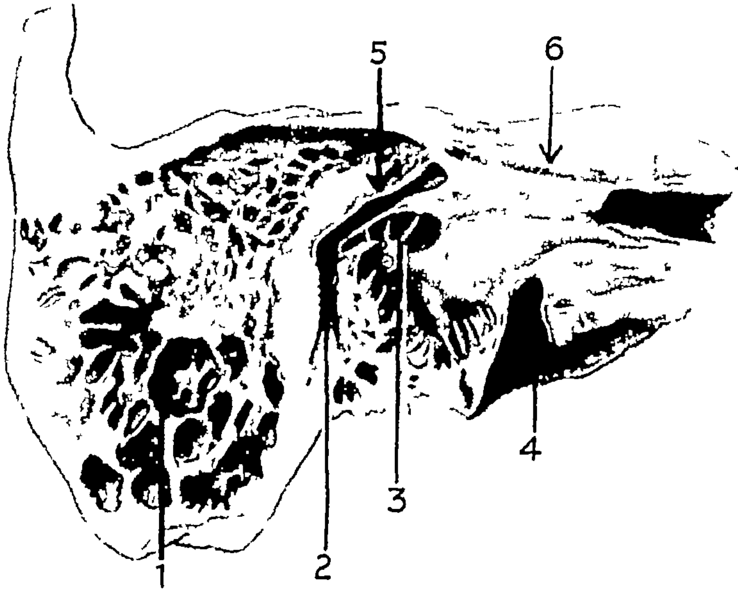


FIG. 110

A cross section of the petromastoid bone showing the facial canal

1, Mastoid air cells 2, Descending part of the facial canal.
3, The stapes 4, The carotid canal
5, The point at which fracture of facial canal usually occurs
6, Groove containing superior petrosal nerve

facial nerve in the transverse part of the facial canal. Paralysis occurring a few days after injury may follow infection (Fig 111) or may be due to the accumulation of blood clot around the nerve or of œdematous swelling of the nerve fibres themselves.



FIG 111

Facial paralysis, the result of suppurative otitis media following a petromastoid injury

Late paralysis developing after many months or a year and not due to infection may occur, and is due to constriction of the nerve fibres by the cicatrisation of healing processes

Prognosis of Facial Paralysis. — Prognosis is difficult to predict in the early stages of paralysis, as little guidance can be obtained from scientific data. However, at least 90 per cent of cases recover, and in my experience only

one case in thirty has failed to do so, and this was due to fracture of the internal auditory and not the facial canal. In this case deafness of the internal-ear type was also present. An incomplete or delayed paralysis is more favourable than a complete and immediate one, since the causative lesion is more likely to be amenable to surgical treatment if the paralysis does not resolve spontaneously. Favourable cases show signs of improvement within two months and usually within two or three weeks (Figs 112 and 113). There is little hope of recovery if no improvement is evident after a year



FIG 112

An example of delayed facial paralysis coming on ten days after injury



FIG 113

Considerable improvement had taken place fourteen days after treatment was started

An incomplete injury of the facial nerve may later lead to facial spasm, a condition equally disabling as, and probably more disfiguring than, paralysis

In many cases of facial nerve injury the chorda tympani nerve is injured, with consequent loss of trigeminal taste (salt, sour, bitter, sweet) on the same side of the tongue Renewal of conduction of taste impulses may be the first sign of recovery, as shown in two of my cases which were observed in detail. Sense of taste first returned and this was followed shortly by improvement in the facial paralysis Another point of interest is that taste is lost on the same side of the tongue but retained on the same side of the palate This is of anatomical rather than of prognostic value, but it does show that the facial nerve is injured distal to

the geniculate ganglion, otherwise the great superficial petrosal nerve, which is the gustatory pathway from the palate, would also be injured

Treatment—*Infection*—As described in the previous chapter, bleeding from an external auditory meatus must always be regarded seriously and treated with great care, otherwise meningitis may result if the dura has been torn. Whenever a fracture opens into a middle ear which is already infected, a radical mastoidectomy should be performed as soon as the patient's general condition will allow. It is unwise to be conservative in these cases and to wait for complications such as spreading osteomyelitis, meningitis or facial paralysis to develop before deciding to operate.

Deafness.—For deafness, little active treatment is possible. It is, in fact, restricted to paracentesis for drainage of blood from the middle ear when the drum is intact and bulging The danger of paracentesis is that infection may be introduced and otitis media result if complete aseptic precautions are not taken

Facial Paralysis—Theoretically, the facial canal should be decompressed in all cases of traumatic facial paralysis whether infection is present or not, particularly when paralysis develops after an interval. In the absence of infection, however, conservative measures give such a high percentage of good results that operative treatment in the early stages should be withheld An exception to this rule is when the facial muscles are rapidly wasting and when electrical reactions of degeneration become evident.

A wire splint, enclosed in fine rubber tubing, crooked over the ear at one end and hooked into the corner of the mouth at the other, should be used immediately and continuously to keep paralysed muscles from being overstretched. Massage and electrical stimulation should be employed every day until full recovery is attained, or until other forms of treatment are decided upon After three months of conservative treatment, if there has been no sign of improvement, the facial canal should be explored and decompressed whatever the electrical reactions of the muscles may be Adequate exploration of the facial canal necessitates radical mastoidectomy When infection has complicated a fracture the fracture lines should be followed and excised as far as anatomical conditions will allow.

INJURIES TO THE BASAL NUCLEI

The hypothalamus [1, 2] consists of a collection of nuclei situated in the brain immediately above the sella turcica in the floor of the

[1] Dott, N M, Le Gros Clark, and Riddoch, G "Hypothalamus" Oliver & Boyd Edin, 1938
[2] Fulton, J F "Physiology of the Nervous System" Oxford University Press, 1938

third ventricle. The nuclei may be divided into three groups: (1) anterior, including the paraventricular and supra-optic group; (2) the middle, including the tuber, lateral, dorsomedial and ventromedial hypothalamic nuclei; and (3) the posterior group, including the posterior hypothalamic nucleus and mammillary bodies. By means of connecting fibres all these nuclei are brought into close communication with each other and with the cortex and optic thalamus

The hypothalamus is the part of the brain which is concerned in regulating the functions of the autonomic nervous system. In its posterior part is situated the centre for control of the sympathetic nervous system Its central and anterior parts are concerned with parasympathetic regulations

The various functions of the hypothalamus have been determined chiefly by animal experiment Stimulation of the posterior hypothalamus produces changes characteristic of sympathetic hyperactivity, such as increase in the heart rate, rise in blood pressure, increased metabolism, dilatation of the pupil and inhibition of the intestinal movements Destruction leads to the opposite effects, including lethargy The tuber is concerned with sweating and with movements of the stomach and intestines. Destruction of the paraventricular nucleus causes hypoglycæmia and destruction of the supra-optic nucleus diabetes insipidus. The whole of the metabolic processes of the body are, in fact, under control of the hypothalamus Many observations made experimentally have been confirmed in man, when the floor of the third ventricle has been accidentally bruised in the removal of pituitary tumours and when degenerations or neoplasms have affected these regions

Injury to the hypothalamus resulting from a violence of the head is by no means rare, as proved by autopsy findings This is not surprising, since fractures of the base tend to converge on the pituitary fossa immediately above which the hypothalamus is situated

The most typical hypothalamic syndromes which occur as a result of head injuries are as follows —

(i) Hyperthermia
(ii) Diabetes insipidus.
(iii) Frohlich's syndrome.
(iv) Acute erosions of the stomach.
(v) Diencephalic epilepsy.
(vi) Hypersomnia.

ULCERATION OF THE ŒSOPHAGUS AND GASTRO-INTESTINAL CANAL

The etiology of gastric and duodenal ulceration has long been a subject of clinical and experimental interest. Cushing[1] was the first to show that lesions in the region of the third ventricle or hypothalamus occasionally resulted in gastro-intestinal ulceration, and it is now becoming evident that hypothalamic influences are of considerable importance in the production of so-called nervous dyspepsias[2]

ACUTE GASTRIC EROSIONS

Acute gastric erosions and perforations of the stomach and œsophagus of neurogenic origin certainly occur, and the following case will show that they may result from injury to the head A youth under my care in the Stockport Infirmary died of acute peritonitis on the twelfth day following a severe injury to the head It was ascertained from his mother that he had not previously suffered from dyspepsia An autopsy examination, at which I was not present and not conducted by my friend Mr Andrew McGill, was made by an outside and inexperienced pathologist at the request of the coroner He came to the conclusion that the youth had died of perforation of the lower end of the œsophagus and fundus of the stomach, consequent on corrosive poisoning. He did not attempt to explain the fact that the mouth and upper end of the œsophagus were not affected At the request of the hospital authorities I was asked to confirm the post-mortem findings. On reopening the abdomen I found definite evidence of recent inflammatory changes in the peritoneal cavity which could not be accounted for by post-mortem autolysis. The stomach and œsophagus were missing, as these had been removed and sent to the county analyst for examination of their contents The brain had been sliced into many pieces and these were found tucked away in the pelvis. These were collected and fixed in a solution of 10 per cent formalin Later, with the aid of Professor Jefferson, the jigsaw puzzle of the brain was fitted together and petechial hæmorrhages were found in the floor of the third ventricle The analyst's report returned negative for corrosive poisoning, and the boy's mother presented me with the specimen of the stomach and œsophagus In this, perforated ulcers were found in the lower end of the œsophagus and upper end of the stomach (Fig 114) Although histological examinations were

[1] Cushing, H "Peptic Ulcers and the Interbrain" *Surg Gyn & Obst*, 1932, **55**, 1
[2] Beattie, J "Hypothalamic Mechanisms" *Canad Med Ass Jour*, 1932, **26**, 400

not made of the walls of the ulcer, the specimen being inadvertently destroyed before this could be done, it is reasonable to presume that the gastric and œsophageal ulceration was neurogenic in origin and the result of the hæmorrhages in the hypothalamus. At no stage in his illness did the youth regain consciousness.

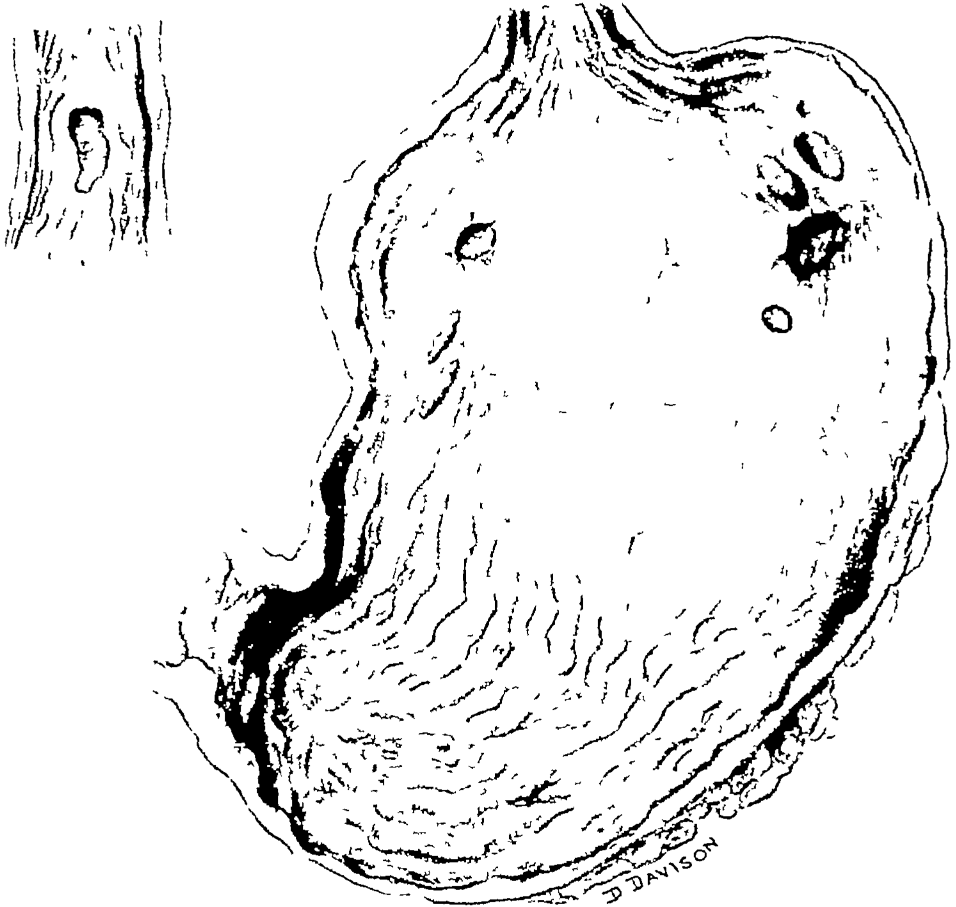


FIG 114

Neurogenic ulcers of the œsophagus and fundus of the stomach resulting from hæmorrhage into the hypothalamus

TRAUMATIC DIABETES INSIPIDUS

Traumatic diabetes insipidus usually declares itself early in convalescence though it may not appear till several months after the injury The outstanding symptom in this condition is insatiable thirst with a desire for water to assuage it In severe cases a patient will drink 1 or 2 pints of water at a time and has to take copious supplies to bed with him as he so often wakens in the night and wants to drink. Fluids taken by mouth are rapidly excreted by the kidneys so that a patient tends to lose weight rather than to become waterlogged. Severe headaches, loss of physical energy and mental depression are commonly

associated with the symptom of thirst One young patient of mine who had been severely concussed and whose skull had been fractured through the pituitary fossa needed at least 12 pints of fluid in the day to satisfy his thirst, and once drank as many as 15 pints. The amount of water he passed was graphically described by his mother as being worthy of a donkey In fact a large bucket was left under the bed for his use as his nocturnal peregrinations used to disturb the whole household. This disturbance of water metabolism was readily controlled by hypodermic injections of 0 5 c c of pituitrin night and morning. In six months after the injury this young man made a complete recovery The tendency in all cases of traumatic diabetes insipidus is towards spontaneous cure. It is useless to restrict the amounts of fluids taken in an attempt to control the diabetes, because this only leads to dehydration and toxæmia Usually injections of pituitrin will keep a patient's thirst under reasonable control until spontaneous cure of the diabetes results Severe and persistent cases may necessitate a total thyroidectomy as suggested by the work of Mahoney and Sheehan [1]

A pathological increase in weight due to excessive deposits of fat occasionally results from a basal injury, as evidenced by the case of a late Professor of Surgery in Manchester who fell from a horse and fractured the base of his skull Following the injury, he became extremely stout, and this stoutness was associated with very definite dispositional changes. Hypersomnia following injury is extremely rare.

Apart from the above symptoms, which afford obvious evidence of damage to the hypothalamus, there are probably many others which also are the result of this type of injury, although they are usually attributed to anxiety or malingering Frequently a patient complains of peculiar sensations of chilliness about the body and abdomen which might be vasomotor in origin. Diencephalic epilepsy [2] probably occurs more frequently than is usually believed. For example, attacks of shivering and sweating for no apparent reason are probably hypothalamic in origin. Emotional outbursts and surges of rage are occasionally seen in the acute stages of cerebral trauma, and these, it may with reason be suggested, are sometimes due to contusion of the optic thalamus.

Whether injury to the caudate and lenticular nuclei can result in Parkinsonism [3] is a subject of great medico-legal importance, and the problem has not yet been finally settled My own opinion is that it can, because a typical syndrome of the disease, which

[1] Mahoney, W, and Sheehan, D "Effect of Total Thyroidectomy upon Experimental Diabetes Insipidus in Dogs" *Amer Jour Physiol*, 1935, **112**, 250
[2] Penfield, W G "Diencephalic Autonomic Epilepsy" *Arch Neur & Psych*, 1929, **22**, 358
[3] Weil, M O, and Oumansky, V "Parkinsonisme Traumatique" *Rev Neurol*, 1937, **67**, 489

medical evidence can prove was not present before the accident, may be discovered in the early stages of convalescence following an injury Unlike the post-encephalic form of the disease it is not a progressive pathological state.

The exact rôle played by the basal ganglia in the complication of acute cerebral trauma is at present obscure, but evidence is accumulating to show that it is an important one and worthy of more detailed investigation.

PULSATING EXOPHTHALMOS

Although over 600 cases of pulsating exophthalmos have been described in the literature, its incidence in any series of head injuries is exceedingly small In the past fifteen years I have seen it on three occasions only and one of these was in America.

No doubt it is its dramatic appearance which accounts for so many cases having been described, and certainly it is because of its distressing symptomology that numerous bold surgical procedures have been used in an attempt to cure it. The underlying cause of the condition is a fistulous opening from the carotid artery into the cavernous sinus either directly or through an aneurysmal sac, arterial blood being blown under relatively great pressure into the venous system of the cavernous sinus with the result that the tributary veins become enormously distended and pulsate with the heart beat

Seventy-five per cent of the cases of pulsating exophthalmos are due to trauma and 25 per cent. to spontaneous rupture of the artery When the result of trauma, the majority are caused by closed fractures of the middle fossa and not by penetrating wounds

The first symptom is a noise in the head which can be heard by the patient and on auscultation. In the early stages the noise may be present only in systole but later becomes continuous with a systolic exaggeration In character the noise may be swishing, banging, beating or clanging, and is worse when the patient is lying down in a quiet room

Usually the second symptom is diplopia consequent on stretching of the ocular nerves as they pass along the wall of the cavernous sinus This is followed by protrusion and pulsation of the eye According to Dandy,[1] this occurs within twenty-four hours in 33 per cent of cases

As the veins of the orbit distend, the eyeball is displaced according to the position of the main ophthalmic veins, and

[1] Dandy, W E "Carotid-cavernous Aneurysms (Pulsating Exophthalmos)" *Zentralbl f Neurochir*, 1937, 2, 77 and 165

as these are usually situated in the upper and inner quadrant of the orbit the displacement is commonly downwards and outwards. Also, the conjunctiva become chemosed, and in serious cases the cornea may ulcerate. Papillitis and blindness are later complications.

The following case, which is still under my care, will illustrate the typical sequence of events in the life-history of a case of pulsating exophthalmos and how the condition may be cured by operative means.

A young Australian soldier was enjoying himself one night in his billet with his friends, and on the way back to his room tripped and fell. He was concussed for a few hours and the next morning was aware of a feeling of fullness in the head. Some days afterwards he was conscious of a noise in his head and later was examined by my colleague Professor Nattrass, who made a diagnosis of a traumatic aneurysm of the internal carotid artery. When transferred to my care his only complaint was of an intermittent throbbing noise in the head. On auscultation over the anterior part of the temporal region on both sides a murmur could be heard which was synchronous with the heart beat and which resembled the clumping of heavy boots on the wooden floor of a large empty hall. Digital compression of the carotid vessels in the neck on the right side caused the murmur to disappear, but not when the vessels on the left side were compressed. At this stage there was a little fullness of the right upper eyelid but no pulsation in the orbital veins. The eyeball was not displaced and did not pulsate. Compression of the carotid vessels for more than a minute caused a feeling of giddiness, thus proving that the collateral circulation and the circle of Willis was inadequate to permit arterialisation of both sides of the brain through one carotid artery.

It was decided, therefore, to compress the vessels digitally in the neck for increasingly lengthening periods until occlusion of the vessels could be maintained for one hour without the development of symptoms.

Whilst this treatment was in progress the patient developed diplopia, but no obvious paralysis of the muscle could be demonstrated. On this evidence of retrogression it was decided to ligature his internal carotid artery in the neck. On the morning of the operation his double vision was much worse, and there was a definite protrusion but no pulsation of the eyeball. The man felt ill and was very worried about himself.

Under intravenous pentothal anæsthesia the internal carotid artery was exposed and an angiogram made by injecting 10 c.c. of thorotrast into the artery.

The X-ray showed that the shadow of the internal carotid artery ceased at the cavernous sinus and that the ophthalmic veins and lateral sinus were clearly filled with thorotrast (Fig. 115) This picture, of course, is what might be expected in view of the pathology of the condition.

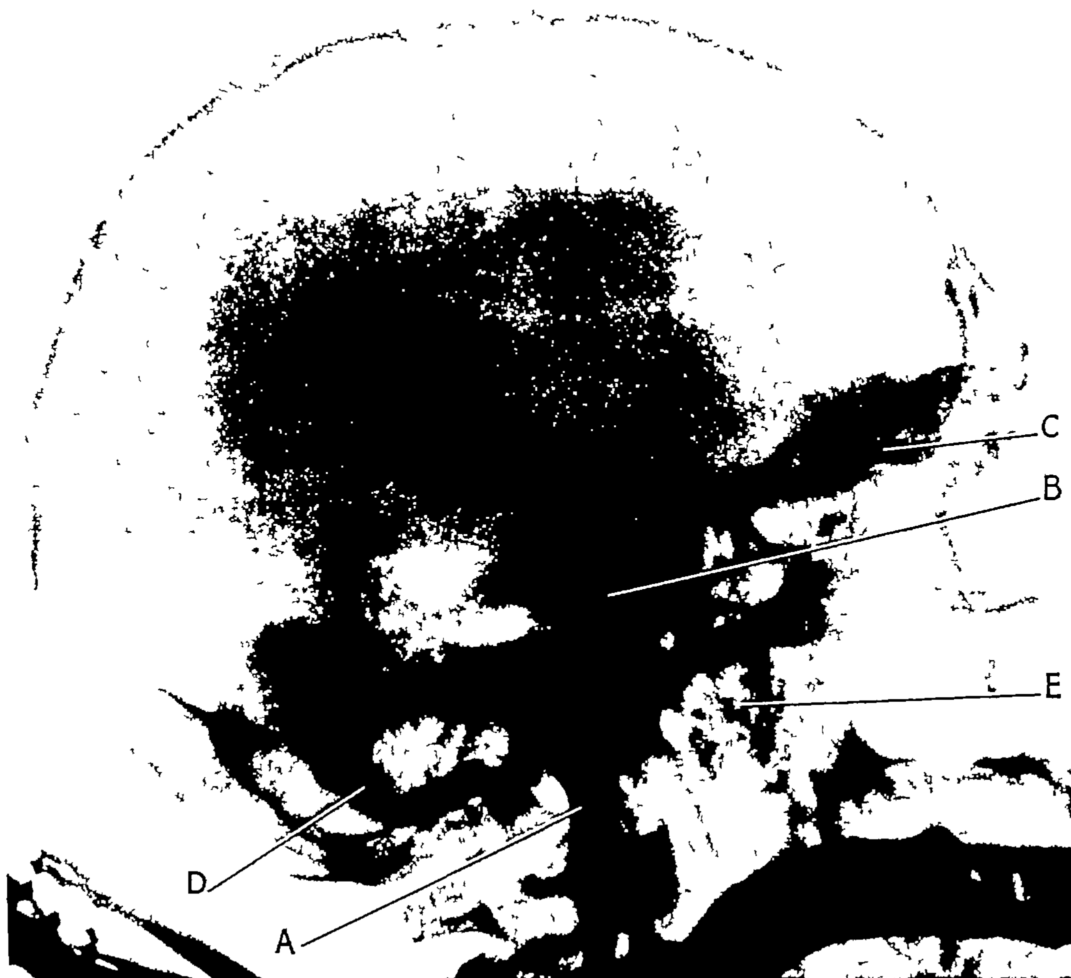


Fig 115

An angiogram depicting the state of affairs in the case of a cavernous arteriovenous aneurysm due to trauma The internal carotid artery A is seen coursing upwards through the neck to end in the cavernous sinus B Normally the S-shaped outline of the intracranial part of the internal carotid shows quite clearly before it breaks up into the branches of the anterior and middle cerebral arteries (see Fig 11) From the cavernous sinus the thorotrast has flowed into the ophthalmic vein C, the lateral sinus D and the pterygoid plexus of veins E It is pulsation and engorgement of the ophthalmic veins which leads to pulsating exophthalmos

The man was then allowed to come round from the anæsthetic until he was conscious and fully co-operative. At this stage I compressed the internal carotid artery between my finger and thumb for twenty minutes and repeated tests as regards muscle power were made on the opposite side of the patient's body. As no neurological signs developed the vessel was ligated firmly

as these are usually situated in the upper and inner quadrant of the orbit the displacement is commonly downwards and outwards Also, the conjunctiva become chemosed, and in serious cases the cornea may ulcerate. Papillitis and blindness are later complications.

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CHAPTER VII

THE SEQUELS OF INJURIES OF THE HEAD

OF the people killed every year on the British roads—and the number is rapidly approaching 9,000—at least 80 per cent die as a result of injuries to the head, as judged by statistics of traumatic deaths in my own hospitals Furthermore, for each fatality due to cerebral causes there are five non-fatal but severe injuries of the head which lead to prolonged morbidity and occasionally to partial or complete incapacity

In this chapter we are concerned only with the late results of cranial and cerebral injury, whether of the open or closed type or whether received under war-time conditions and by accident in civilian life That these late results or sequels are multitudinous in kind is not surprising, since the mechanism of most injuries to the head is such that the whole brain is subjected to the injuring force and suffers accordingly. Neurones or nerve cells, neuroglia or interstitial tissue, blood vessels and their controlling mechanisms, meninges and cerebrospinal apparatus are all damaged to some extent, as well as the scalp and the skull.

The most common sequels which follow severe trauma can be classified thus.—

I Of the Brain.

(*a*) *Those due to Diffuse Injuries.*

Common Symptoms.

1. Headaches
2. Dizziness.
3. Insomnia
4. Changes in Disposition.
5. Mental sluggishness in adults.
6. Intellectual retardment in children.
7. Anxiety neurosis.

Rare Symptoms:

8. Hysteria
9. Psychoneurosis.
10. Psychosis.

with stout silk and the wound left open for another fifteen minutes to make sure that hemiplegia did not develop.

The noise in the head ceased at the moment of compression, and the man's mental attitude changed dramatically to one of optimism. For a month after operation the patient was left in bed with his head low and with the foot of the bed raised for the first two weeks.

IV. Of the Scalp.

1. Loss of tissue with deformity
2. Loss of hair.
3. Neuralgia

POST-TRAUMATIC EPILEPSY

A comprehensive definition of epilepsy has not yet been produced and probably will not be forthcoming until the actual nature of the condition is known.

As Kinnier Wilson[1] has pointed out, it is obviously impossible to embrace all its varieties in a single clinical formula, particularly as some are the result of inhibited rather than of excessive nervous activity The modern tendency is to look upon epileptic phenomena as being the result of an unusual discharge of the neurones either in the form of an overcharge, or of an inhibition, or of a change in rhythm. Whether all epileptic states are essentially the result of pathological processes or of congenital abnormalities within or on the surface of the brain tissue is not known, but what is certain is that structural changes are now being reported rather more frequently than hitherto. This presumably is due to the advance in neuropathology rather than to any change in the etiology of epilepsy itself

With reason it may be postulated that before epileptic phenomena can develop, an epileptic predisposition in some part of the brain tissue occurs which, at intervals, becomes activated by physico-chemical changes within the cells themselves, possibly consequent on circulatory and general metabolic influences.

From clinical and experimental observations it has been clearly established that some brains will go into a state of epilepsy much more easily than others. When epilepsy follows minor forms of closed injury it is believed that the condition has merely been precipitated and not actually caused by the injury ; in other words, an epileptic predisposition was already present before the accident On the other hand, certain forms of injury, such as penetrating wounds associated with sepsis, can lead to epilepsy whether there was a previous tendency in the brain to epilepsy or not.

From the therapeutic point of view it is important to realise that an epileptogenic focus does not necessarily correspond to a pathological focus or to the track of a wound. In fact, wherever a structural change due to trauma may be situated, an epileptic attack may start either with psychical, sensory, motor or autonomic phenomena So-called major and minor attacks are

[1] Kinnier Wilson, S A "Neurology" London, 1940

(*b*) *Those due to Local Destruction of Brain Tissue by Contusions or Lacerations*

1. Spastic paralysis
2. Aphasia—motor and sensory
3. Hemianopia.
4. Epilepsy—focal and general.
5. Sensory loss of discriminative type
6. Diplopia due to injury of the ocular nuclei within the brain stem
7. Intellectual impairment due to bilateral contusion of the frontal lobes
8. Diabetes insipidus, adiposis genitalis and ulcers of the stomach due to injuries of the hypothalamus
9. Hydrocephalus
10. Delayed apoplexy

II. Of the Cranial Nerves.

1. Loss of smell and taste
2. Loss of vision
3. Diplopia
4. Sensory loss in the trigeminal field
5. Facial paralysis.
6. Deafness due to contusion of eighth nerve
7. Nerves nine to twelve are rarely damaged

III. Of the Skull.

(*a*) *Closed Injuries*

1. Compression of brain due to depressed fragments
2. Dizziness due to involvement of the labyrinth
3. Deafness—of middle ear type.
   Deafness—of internal ear type.
4. Meningocele

(*b*) *Compound Injuries*

1. Same as in closed type.
2. Infection
   Chronic periostitis
   Chronic osteomyelitis
   Subdural abscess
   Meningitis—focal or generalised.
   Localised intracerebral abscess
3. Defects in the skull
4. Cerebral fungus.
5. Cerebrospinal fluid fistula—ear, nose, vault.

5 Œdema
6 Hydrocephalus
7. Septic encephalitis

II. Delayed Epilepsy (*occurring at any time up to three months*).—This may be due to :—

1. Healing processes
2 Pathological complications

III. Late Epilepsy (*occurring at any time after three months*).—This may be due to :—

1. Scars :
   (*a*) Cerebral
   (*b*) Meningocerebral.
2. Cysts, including porencephaly.
3 Abscesses
4. Foreign bodies
5. Chronic subdural hæmatomata.
6 Traumatic aneurysms.
7. Aerocele
8 Meningitis serosa circumscripta.
9 Degenerative encephalitis.

As the conditions mentioned in the first two groups have been discussed in previous chapters it is the third group with which we are particularly concerned here, and each pathological state will be described separately.

Brain Scars (Cerebral and Meningocerebral).—Brain scars are believed to be the most common cause of epilepsy. As these most frequently result from penetrating wounds, and particularly those complicated by the processes of infection, the incidence of epilepsy is greater in penetrating than in non-penetrating types of injury (Fig. 116). Of all the conditions likely to lead to epilepsy, the attachment of the cortex of the brain to the overlying skin, consequent on superimposed calvarial and dural defects, is the most important A scar attached to the dura occurs as a greyish gelatinous mass of tough and resilient consistency. Histologically a scar is composed of a mixture of fibrous and glial tissue in the meshwork of which are enclosed areas of altered brain tissue. The following is a résumé of a report given by Professor Shaw on a scar which I excised from the frontal pole of a young boy's brain :—

The specimen consists of an oval piece of brain tissue 4 × 4 × 2 2 cm One longer surface is covered by vascular membranes, the other is covered by fragmented hæmorrhagic brain tissue and is presumably the line of excision There is a V-shaped depression 2 cm long and 0 8 cm deep on

essentially due to the same physio-pathological processes, the difference being purely the extent of the cerebral disturbances In minor attacks the abnormal neuronal discharges remain confined to a circumscribed area of the brain, whereas in major attacks the whole brain may be affected. Whether a patient becomes unconscious depends largely on the part of the brain concerned but also to some extent on the amount of brain tissue involved. Though epilepsy may not necessarily be caused by a progressive pathological state in the same sense as a degeneration or new growth, repeated attacks may lead to structural changes as a result of the trauma inflicted at convulsive episodes

PATHOLOGY

An injury to the head does not produce a static pathological lesion within the brain. From the moment of violence, through the stages of phagocytosis to the final consolidation of a wound, or during the phases of possible secondary degeneration or inflammation, the state of the brain is constantly changing It will be realised, therefore, that epilepsy may result from one or a combination of numerous pathological possibilities It is surprising, however, how rarely actual solution of continuity of the brain tissue leads to an epileptic seizure For example, I had a man under my care recently who had been struck over the head with an axe, causing a deep lacerated wound of the motor cortex, and at no time up to his death many days later did he suffer from an epileptic seizure. Epilepsy also rarely occurs during the processes of repair The condition usually develops some time after repair has been completed and is the result of scar tissue or of some pathological sequel such as abscess

I. Immediate Epilepsy (*occurring whilst the patient is still unconscious*) —This may be due to —

1 Contusion
2. Laceration.
3 Hæmorrhage.
   (*a*) Intraventricular.
   (*b*) Intracerebral
   (*c*) Subpial
   (*d*) Subarachnoid.
   (*e*) Subdural
   (*f*) Extradural
4. Bone fragments :
   (*a*) Depressed and causing compression
   (*b*) Penetrating and irritative.

external surface, and a section through this shows atrophy and in places disappearance of the cortical grey matter which is replaced by a linear-shaped, firm pearly tissue, like scar tissue, extending into the cerebral tissue to a depth of 1 5 cm

"Microscopically the surface of the cerebral cortex is covered by a thick layer of collagen tissue of varying grades of maturity in which are a number of arterioles and large venules, and also a dural sinus in which is an arachnoid villus with its crest of secretory cells This collagen tissue therefore represents scarring of the dura mater and also, probably, of the pia-arachnoid as well, all of which is adherent to the cortex At the site of the depression the scarred membranes become continuous with a dense scar consisting, as special staining shows, of collagen exhibiting extreme hyalinosis and containing hæmosiderin, this passes down into the white matter and corresponds to the linear scar seen in the gross

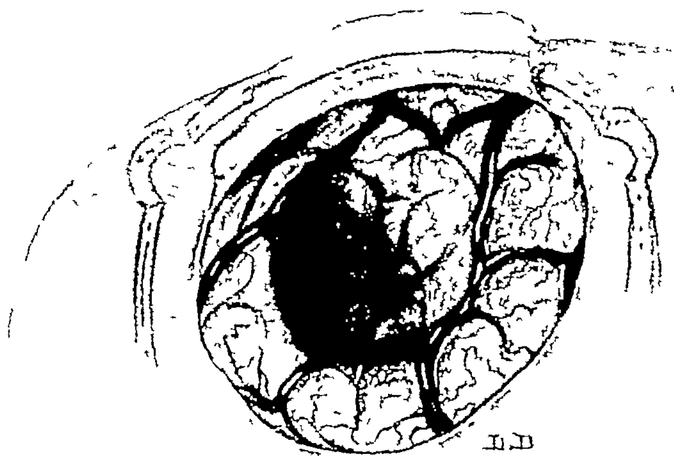


FIG 117
The dimple of a cerebral scar

"Grey matter with its constituent neurons persists on either side of the depression, and for some distance on each side there is complete *disappearance of the grey matter*, the gap being filled by *extensive gliosis*, which extends down into the white matter around the collagen scar In the gliosis are several small irregularly-shaped cystic areas

"The appearances are indicative of injury, resulting in destruction of the grey matter, followed by healing due to scarring of the meninges and replacement gliosis The collagen scar deep in the white matter is probably derived from mesoblastic elements of the meninges driven into the cerebral tissue at the time of the trauma"

When not attached to the dura a scar is seen as a depression on the surface of the brain, the floor of which is composed of shrunken gyri stained by yellow pigment [1] (Fig 117).

[1] Elvidge, A R "Brock's Injuries of the Skull, Brain and Spinal Cord," chapter ii Baillière, Tindall & Cox London, 1940

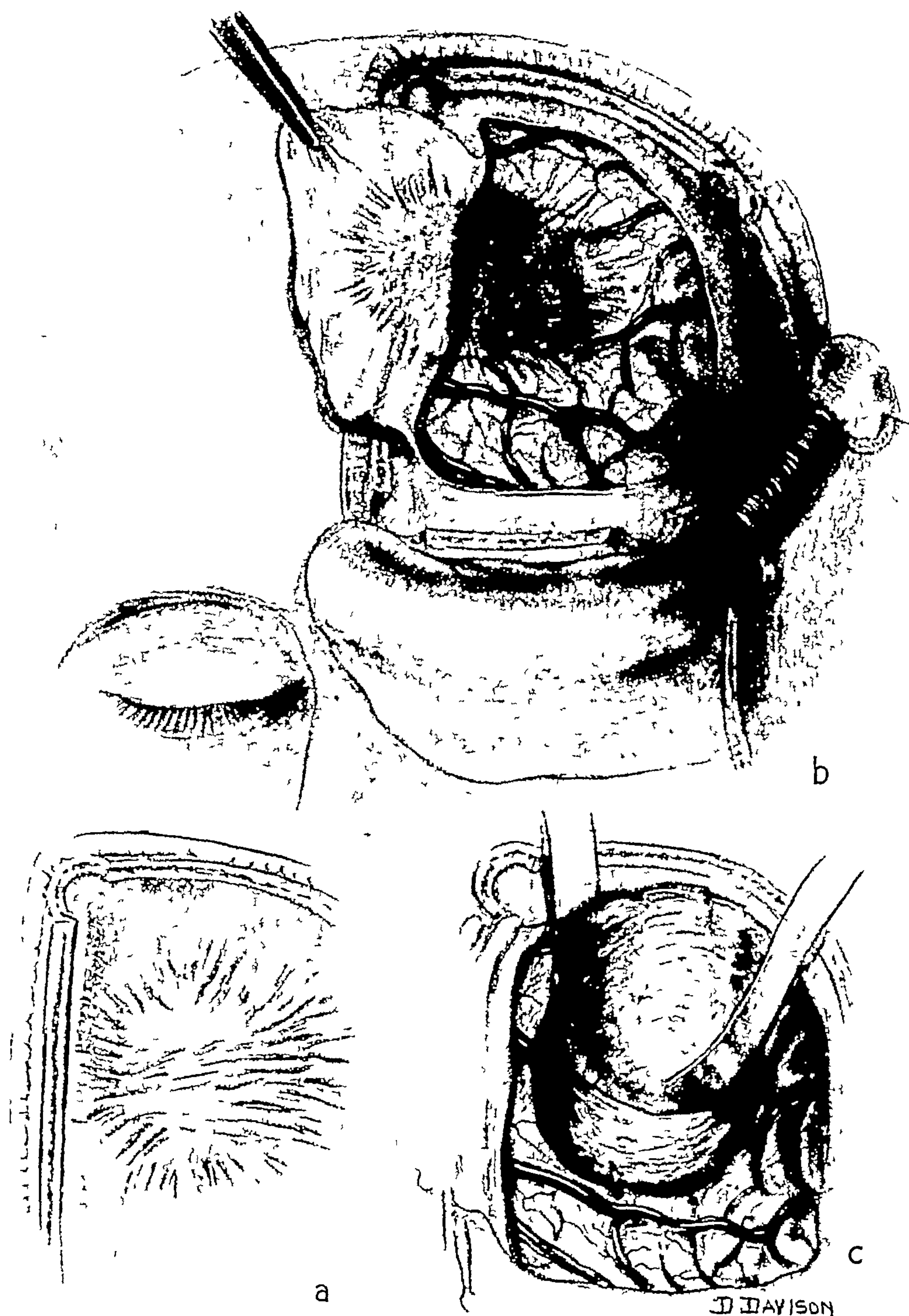


Fig 116

A meningo-cerebral scar

a, The appearance of the dura mater
b, Reflection of the dura exposing the cerebral scar
c, Removal of a cone of cerebral tissue containing the fibro glial cicatrix.

they develop hard fibrous capsules which irritate the surrounding brain

Meningitis serosa circumscripta is the name given to small loculations of cerebrospinal fluid within meningeal adhesions, which result either from direct contusion or to reactions aroused in the lining cells by the irritation of blood in the subarachnoid spaces

Vascular Anomalies.—In particular, aneurysmal varices or varicose aneurysms are occasionally seen on the surface of the brain when cranial explorations are being made for the treatment of post-traumatic epilepsy Whether these conditions may result from a blow on the head is extremely doubtful, although theoretically they can. In the large majority of cases they are congenital in origin, and it is known that such conditions may lead to epilepsy at any time in life, irrespective of whether the head is injured or not It must be admitted, however, that cirsoid aneurysms on the cortex may exist and not produce epileptic phenomena until the adjacent brain tissue has been altered by contusion or other traumatic processes.

Aerocele.—As a result of compound fractures of the skull involving the dura, particularly when the paranasal air sinuses are concerned, air may be sucked into the intracranial cavity, to collect either in the ventricles or in the subarachnoid spaces. On occasions a lacerated surface of the brain becomes attached to the margins of a dural fistula, when air may be blown or sucked in the cerebral tissue itself to produce an air cyst This may increase in size and behave like any other expanding lesion and produce epilepsy, usually of the frontal lobe type, for the good reason that the frontal lobes are usually involved. All that is necessary in these cases in the way of treatment is to repair the causative fistula and to aspirate the cyst.

COMMENTARY

Neoplasms, such as meningiomata, are a common cause of epilepsy, being occasionally found when there is a definite history of severe injury to the head. What part trauma may play in the production of cerebral neoplasia is not known Until this problem has been satisfactorily settled on a scientific basis it is best to regard new growths following head injuries as coincidental On the other hand, patients who have had a partial removal of a cerebral tumour and who later receive an injury to their heads, commonly retrogress very rapidly and in a way which suggests that trauma may influence the rate of growth of a neoplasm when once it is established

Foreign Bodies.—A foreign body buried deeply beneath the cortex of the brain is in itself not considered to be an important causative factor in the development of post-traumatic epilepsy. What is important is the track of its wound with its resulting cortical scar. A fragment of bone which penetrates the dura and which is left spiking the cortex is far more likely to cause epilepsy than when it passes into the deeper cerebral layers, because repeated pulsations of the brain against it are apt to lead to extensive surface scarring. In recent years, owing to enemy action, numbers of patients have received penetrating wounds of the head and have been left with metal fragments embedded in their brain. My opinion is that deeply embedded fragments not removed at the primary débridement should not be disturbed if they are not causing obvious inflammatory complications, as their removal is most unlikely to lessen the chances of subsequent epilepsy. On the other hand, I believe that superficially placed and easily accessible fragments should be removed as a prophylactic measure. When epilepsy has already developed, bony or metal fragments may or may not have to be removed according to whether or not they lie within the epileptogenic focus.

Cysts of the Brain and Porencephaly.—Porencephaly implies that there is a hole in the brain, and in the true congenital variety ventricles and the subarachnoid spaces are in wide communication. In acquired cases the condition is usually found in the form of a cystic cavity lined with flattened cells or condensed brain tissue, communicating only with a ventricle or with the subarachnoid spaces. Porencephalic cysts often start at the junction of the white and grey matter. It is believed that they result from destructive vascular processes such as hæmorrhage, thrombosis and embolism. Cysts containing yellowish albuminous fluid within the brain tissue or immediately beneath the pia and not in communication with the ventricles or surface spaces are occasionally seen, these also are due to softening following destructive vascular processes.

Chronic Subdural Hæmatomata.—These are often the cause of epileptic seizures, but whether they produce this state by compression or through associated thrombotic changes in the underlying brain tissue is not known. Probably they do so by compression, because drainage of a hæmatoma is usually all that is necessary to rectify the epileptic state.

Abscesses of the Brain are almost invariably the result of penetrating wounds, and occasionally fragments of bone or pieces of metal or cloth are found within their cavities. They are not of common occurrence, but on the other hand they are particularly liable to lead to epilepsy, and this no doubt is due to the fact that

TABLE II

INCIDENCE OF EPILEPSY IN HEAD INJURIES WITH PENETRATION OF DURA

Author	Year	Number of Cases Injured	Percentage Epilepsy	Remarks
Behague [2]	1922		Over 12	Periods of onset Shortest—under 3 months Longest—18 months
Rawlings [4]	1922	228	32 8	Gunshot wounds
Steinthal and Nagel [6]	1926	348	31 6	
Wagstaffe [7]	1928	176	18 7	Gunshot wounds with penetration of dura
Credner [9]	1930	1,234	49 5	Periods of onset Shortest—within 1 year Longest—10 years
Ascroft [13]	1941	129	45 0	Gunshot wounds
Brun (quoted by Steinthal)		21	33 3	Gunshot wounds with contusion of motor area

The figures of Ascroft may be accepted as a very reliable indication of the results of gunshot wounds of the head, and it is very interesting to note that with non-penetration of the dura this author found that the incidence of epilepsy is as high as 23 per cent. No doubt the manner in which war-time wounds are inflicted—that is, by small objects travelling at great speed and leading to penetration—accounts for the disturbingly high figures tabulated above.

Under peace-time conditions the mode of injury is different. This is usually of the blunt type, the head being struck by a broad surface so that penetrations and septic complications are uncommon

[1] Allen, D P, Sanford, L H, and Dolley, D H "Traumatic Defects of the Skull Their Relation to Epilepsy A Clinical and Experimental Study of their Repair" *Bost Med & Surg Jour*, 1906, 1, 396

[2] Behague, P "Etude sur l Epilepsie traumatique" Thèse de Paris (Arnette, Paris, Editeur) 1919 Analysée par Feindel *Rev Neurol*, 1920, **36**, 88 "Characteristics and Treatment of Traumatic Epilepsy" Questions Neurologique d'Actualité Paris Page 475

[3] Sargent, P "Some Observations on Epilepsy" *Brain*, 1921, **44**, 312

[4] Rawlings, L B "The Remote Effects of Gunshot Wounds of the Head" *Brit Jour Surg*, 1922, **10**, 93

[5] Turner, W A "Epilepsy and Gunshot Wounds of the Head" *Jour Neur & Psychopath*, 1922, **3**, 309

[6] Steinthal, K, and Nagel, H "The Capacity for Work after Gunshot Wounds of the Brain, with Special Reference to Traumatic Epilepsy" *Beitr z Klin Chir*, 1926, **137**, 361

[7] Wagstaffe, W W "The Incidence of Traumatic Epilepsy after Gunshot Wounds of the Head" *Lancet*, 1928, **218**, 861

[8] Steinthal, K "Epilepsy, in Particular Traumatic Epilepsy and the Results of Surgical Treatment" *Ergeb d chir u Orth*, 1929, **22**, 222

[9] Credner, L "Clinical and Social Effects of Lesions of the Brain" *Zeitschr f d ges Neurol*, 1930, **126**, 721

[10] Feinberg "Epilepsie und Trauma" 1934

[11] Marburg, O "Die Traumatischen Erkrankungen des Gehirns und Ruckenmarks" *Handb der Neur*, O Bumke und O Foerster, 1936, 2 J Springer, Berlin

[12] Elvidge, A R "Post traumatic, Convulsive and Allied States" 'Brock's Injuries of the Skull, Brain and Spinal Cord" Baillière, Tindall & Cox London, 1940

[13] Ascroft, P B "Traumatic Epilepsy after Gunshot Wounds of the Head" *Brit Med Jour*, May 17, 1941

On rare occasions a cerebral tumour may be the cause of accident, as is well illustrated by the following case. Some years ago I was asked to see a young girl who had received a severe injury to her head, having been knocked from her pedal cycle by a bus The bus driver stated that the girl was riding unsteadily on her cycle, and that she swerved into him when he was about to pass. When I first saw the girl she was conscious and showed all the signs of a cerebellar tumour, including papillœdema This diagnosis was finally confirmed at autopsy, and inquiry proved that there was evidence of cerebellar dysfunction previous to the accident.

Degenerative and progressive encephalopathies associated with epilepsy may follow injury, and more will be said on this subject later.

THE INCIDENCE OF POST-TRAUMATIC EPILEPSY

As the following tables will show, the incidence of post-traumatic epilepsy varies considerably according to whether the causative injury was inflicted under peace or war time conditions (Tables I, II and III)

TABLE I

INCIDENCE OF EPILEPSY IN HEAD INJURIES OF ALL TYPES

Author	Year	Number of Cases Injured	Percentage Epilepsy	Shortest Period before Onset	Longest Period before Onset	Remarks.
Allen Sanford and Dolley [1]	1906	571	4·3			Gunshot wounds Franco-Prussian War
Behague [2]	1919	3,523	12·1			Gunshot wounds.
Sargent [3]	1921	18 000	4·5			Gunshot wounds Ministry of Pensions
Rawlings [4]	1922	452	25·0			Gunshot wounds
Allen (quoted by Turner) [5]	1923	167	13·7			Gunshot wounds. American Civil War
Steinthal and Nagel [6]	1926	639	28·0		Several years	
Wagstaffe [7]	1928	377	9·8	2 months	7½ years	Gunshot wounds.
Steinthal [8]	1929	531	0·2	18 years	18 "	Civilian hospital Only one case.
Credner [9]	1930	1 990	38·2	Under 1 year	10 "	1 103 of these cases were observed for over 5 years
Feinberg [10]	1934	47 130	0·1			
Marburg [11]	1936		25·0	1 month	23 years	Analysis of several authors.
Elvidge [12]	1940	362	1·03	Three-quarters of an hour	6 months	Civilian hospital Cases observed for short period only
Ascroft [13]	1941	317	34·0			Gunshot wounds. 1914 18
Krause and Schuman (quoted by Marburg)			4·73 to 26·7			War injuries
Brun (quoted by Steinthal)		470	5·3			War time skull fractures.
Reichman (quoted by Steinthal)		603	0·5			Civilian hospital.

1 13—*References on page 227*

Clinical Features.—Clinically an epileptic seizure may be divided into the following phases :—

1. Prodrome.
2. Aura.
3. Content, or fit proper.
4. Sequels.

By *Prodrome* is meant such symptoms and signs as may occur at varying intervals of hours or days before an attack, and these may be motor, sensory, autonomic or psychical in character. Occasionally a seizure is heralded by a change in mood, associated with a feeling of malaise and a fullness in the head. Prodromes, however, are by no means common and are often so unobtrusive as to be unrecognisable except to the most intimate relatives Moreover they are of little diagnostic or surgical value, as they rarely shed any light on the focus or origin of the fit or on the nature of the lesion which causes it.

Aura is the name given to those sensations or motor activities which immediately precede the major event of a seizure and which therefore indicate the part of the brain where the epileptic fit concerned originates. From the surgical point of view an aura is the most important event in any seizure. In fact nothing of value surgically is known about a fit until the absence or presence of an aura has been ascertained, and if present, what its precise character may be.

A fit starting in the autonomic system, possibly in the hypothalamus, usually declares itself by a feeling of uneasiness in the abdomen and by vasomotor changes as shown by circumoral pallor. Sensations of tingling, pins and needles and numbness are referable to the sensory cortex in the post-Rolandic region and can be localised with considerable accuracy. This also applies to motor phenomena when they occur in an orderly manner and slowly enough to permit of visual analysis. Such attacks are usually known as Jacksonian epilepsy. Seizures starting in the occipital lobes are heralded by simple hallucinations of light Formed images probably indicate that the visuo-psychic areas anterior to the calcarine fissure are at fault Buzzing in the ears or other hallucinations of noise are referable to the temporal lobes of the brain. A rather strange condition also referable to the temporal lobes is the so-called "dream state" In this condition the patient is not unconscious, but is far away from his surroundings and feels that he is a witness of some other world, either of the past or the future. The auras most difficult to analyse and to localise are those which are thought to originate from the frontal areas of the hemispheres of the brain. This

As few statements have been made on the incidence of epilepsy following this type of injury, I accepted Professor Jefferson's suggestion to make a follow-up of a series of cases of head trauma treated in the Manchester and district hospitals. It was decided to circularise 1,000 cases whose injuries had occurred not less than five years before. This long interval was chosen so that patients developing epilepsy late after an injury would not be omitted from the final figures, which of course was an important consideration, as post-traumatic epilepsy may develop at any time up to the patient's death In fact I have a record of seizures occurring twenty years after a fall from the top of a tram. In this case local damage was done to the brain, the epileptogenic focus corresponding with the site of the cerebral scar which was in the parietal lobe

Owing to evacuation and other war-time conditions, 430 only of 1,000 cases were traceable. This, of course, is an obvious imperfection in the compilation of any statistical survey, but none the less the figures given below are of value, particularly as I had opportunity to interview and examine every patient suspected of epilepsy and in many cases to obtain X-rays of their skulls.[1]

TABLE III

INCIDENCE OF POST-TRAUMATIC EPILEPSY FOLLOWING INJURIES OF THE HEAD OF THE BLUNT TYPE IN PEACE TIME

Total number of cases injured	430
Total number of cases developing epilepsy	11
Percentage occurrence of epilepsy	2 5

Sex	Date of Injury	Age at Accident	Severity of Accident	Fracture of Skull at Injury	Present Evidence of Fracture.	Time of Onset of First Fit after Accident.	Number of Fits.	Type of Fit.	Neurological Signs of Residual Injury	Capacity to Work.	Remarks.
F	1931	4 years	Moderate	?	No	2 years	3	Petit mal	Nil	Lost one post because of fit while at work	A healthy looking but nervous girl now working as a machinist.
M.	1930	3 weeks	Slight	?	No	Few hours	Many	Petit mal	"	Doing well at school	Parents worried as to what is going to happen to him in the future
M	1931	47 years	Severe	Yes	Yes	7 months	"	Convulsive seizures and unconsciousness	"	Has not worked since	Wife says he is extremely ill-tempered.
M	1930	21 "	"	?	No	15 weeks	"	Sensory Once unconscious	"	Can only do light work	Still feels very ill.
M	1934	23 "	"	Yes	Yes	4 years	Several	Grand mal	"	Is a plumber on full time work	Often works on scaffoldings.
M	1934	18 "	"	?		[illegible] weeks	"	Petit mal	"	Architect	Discharged from army because of psychoneurosis and epilepsy
M.	1931	23 "	Moderate	Yes		2 months	Many	Convulsive seizures and unconsciousness	"	Full work in warehouse but needs concessions	Discharged from army because of fits.
M.	1932	[illegible]6	Slight	"	No	Several years	3	Grand mal	"	Full work as plumber	General health excellent.
M	1931	27 "	Severe	"	"	Within 1 year	Many	Convulsive seizures and unconsciousness	"	Has not worked since	Sits about house most of the day
M.	1931	[illegible]0	"	"	No.	3 yrs 9 mths.	"		"	Full time gardener	Still has frequent and severe headaches.
M.	1933	23 "	"	"	"	1½ years	"	"	"	Has lost employment because of fits	Is an A.R.P. warden.

NOTE.—There were no open head injuries in this group.

[1] This work was made possible by a grant from the Dickinson Scholarship Fund, M R I

The Fit Proper and Sequels.—There are two types of fit—major and minor.

In a major fit large areas of the brain are involved in a cerebral disturbance which leads to profound unconsciousness and to a sequence of phenomena that have been so well described by other writers that little need be said about them here. According to Gowers there is an aura, usually of an indefinable sensation in the epigastrium, in 50 per cent. of fits of the major type In a typical attack a patient falls unconscious to the ground uttering a guttural cry, and goes through succeeding stages of tonic and clonic convulsions, in which he may bite his tongue, froth at the mouth and pass water. He may recover rapidly and carry on as though nothing had happened, or he may fall into a deep sleep and be incapacitated by sickness and headache for days. Also, exhaustion may lead to prolonged paralysis of muscle groups of a limb or limbs.

There are two types of minor fit: petit mal and so-called focal or Jacksonian epilepsy.

Petit mal consists of momentary loss of consciousness or of a disturbance of consciousness short of loss. Typically, a patient will stop what he is doing and stare blankly in front of him for a few moments and then resume in normal sequence what he was doing when the attack intervened

The essential feature of a focal fit is that a cerebral disturbance begins in a circumscribed focus of the brain tissue and remains there or travels to other parts of the brain so slowly and in such an orderly anatomical manner that the "march" of the fit can easily be observed The actual character of a fit depends on the part of the brain concerned and thus may be motor, sensory, including hallucinations of the special senses, or psychical.

Apart from the more stereotyped kinds of epilepsy, there are variants with special features which necessitate particular emphasis, as they occasionally lead to social improprieties, or may even bring a patient within the ambit of the law. Immediately following the major event of an epileptic seizure a patient may enter a state known as "Post-epileptic Automatism" In this condition a patient has full control of his muscle power and co-ordination, and, apparently, is fully conscious. He is not, however, in full control of his judgment, and may perform silly, vulgar, or even dangerous acts without being aware of what has happened when finally his automatic state disappears. When fits are of a psychical character all kinds of complicated hallucinations may be experienced which may lead to or be associated with emotional outbursts or acts of violence known as brainstorms. One of my patients, a married man, suffering from such attacks, would waken in the night with the belief that his late fiancée was

difficulty is due partly to the fact that frontal auras are so rapid that they cannot be followed by clinical methods, and partly because localisation of function, as we know it, may not exist in the frontal association area. Certainly none has been discovered up to the present time.

The sudden cry of a patient about to enter a major fit probably means no more than that the cerebral disturbances are crossing the lower part of the motor cortex and not that the fit has actually started in the cry centre, wherever this might be.

Since Hughlings Jackson's work [1] on cortical localisation was confirmed experimentally by Ferrier [2] in Britain and by Fritsch [3]

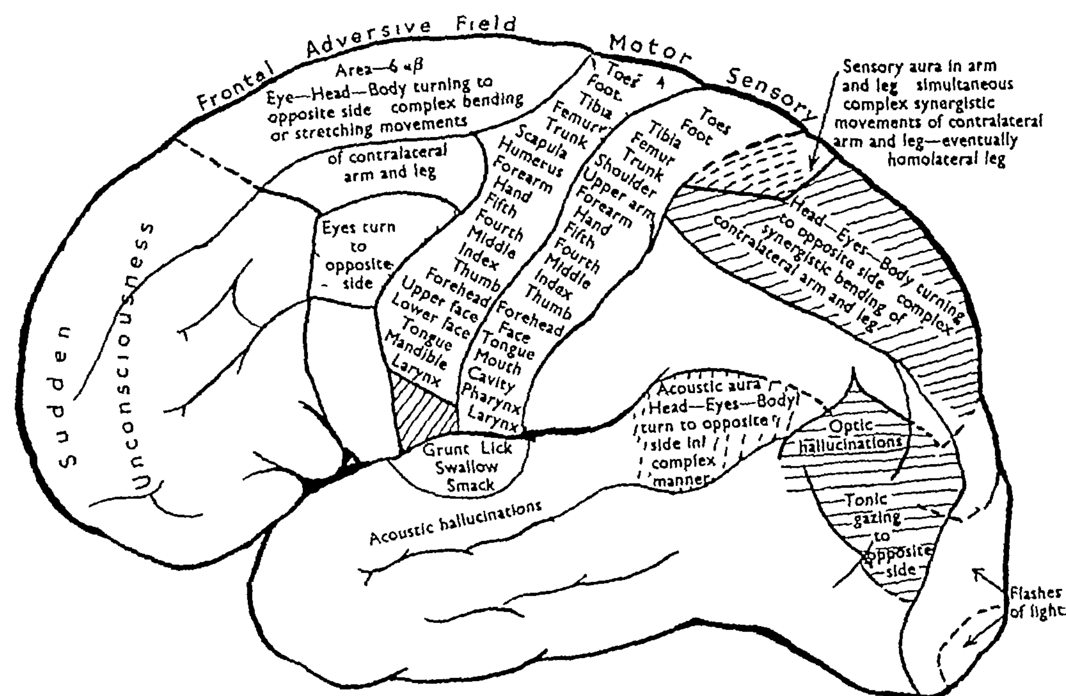


Fig 118
The epileptogenic cortical areas in the human brain

and Hitzig [4] in Germany, every part of the cerebral cortex accessible to the surgeon has either been stimulated by electrical currents or surgically excised. Much work on this subject has been carried out by Foerster [5] and Penfield,[6] and the accompanying diagram embraces the most recent conceptions of the localisation of epileptic auras (Fig. 118).

[1] Hughlings Jackson, J "Selected Writings of John Hughlings Jackson" London, 1931
[2] Ferrier, D "Experimental Researches in Cerebral Physiology and Pathology" West Riding Lunatic Asylum Med Rep, 1873, **3**, 1-50 "The Croonian Lecture Experiments on the Brain of Monkeys (Second Series)" *Philos Trans*, 1875, **165**, 433 488
[3] Fritsch, G, and Hitzig, E "Ueber die elektrische Erregbarkeit des Grosshirns" *Arch. Anat Physiol wiss Med*, 1870, **37**, 300
[4] Hitzig, E "Untersuchungen uber das Gehirn" Berlin A Hirschwald 1874, **6**, 276
[5] Foerster, O and Penfield, W "Epilepsy and the Convulsive State," chapter xix. Baltimore, 1931 *Zeitschr f d ges Neur u Psychiat*, 1930, **30**, 475
[6] Penfield, W, and Gage, L "Cerebral Localisation of Epileptic Manifestations." *Arch. Neur and Psych*, 1933, **30**, 709

By suitable placing of the electrodes, electroencephalography can indicate the site of the brain in which the electrical discharge originates. In some cases it can be shown that epileptic attacks may start from a circumscribed area on one side of the brain,

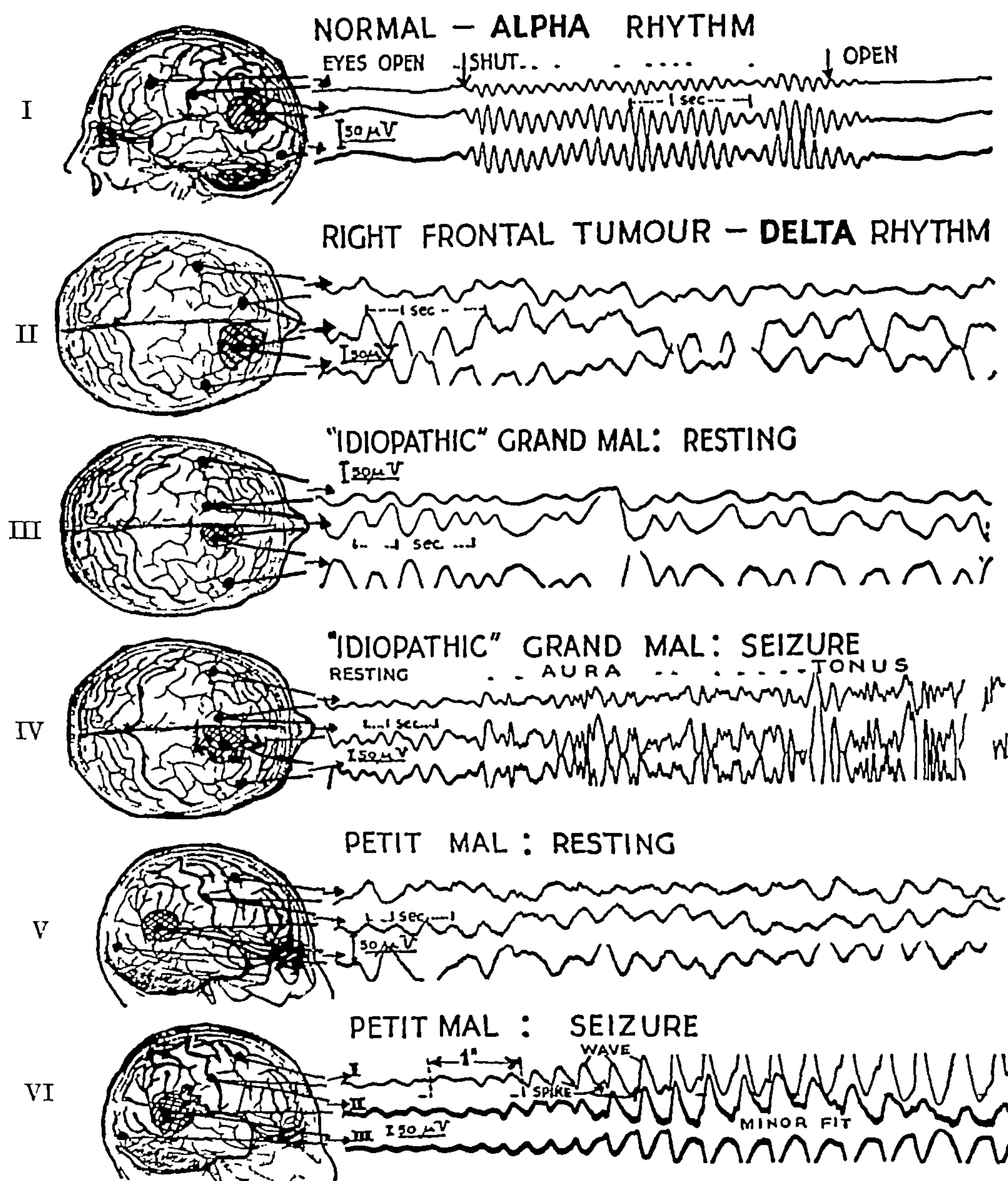


FIG 119

The diagrams on the left show the positions of the leads and of the discharging foci (shaded), the tracings on the right show the corresponding changes of electrical potential

(*From Brain's "Recent Advances in Neurology" Electroencephalograms kindly lent by Dr W Grey Walter*)

I, Normal alpha rhythm.
II, Delta rhythm in a patient with a right frontal tumour
III, Resting rhythm in a patient with "idiopathic" grand mal
IV, Seizure rhythm in a patient with "idiopathic" grand mal
V, Resting rhythm in a patient with petit mal
VI, Seizure rhythm in a patient with petit mal

entreating him to reconsider his decision and to go home with her. This would start a struggle in which he would attempt to evict her from his bedroom in the fear that his wife would waken and discover his embarrassment. So violent would the struggle become that it would end on the floor, and his wife's description of the attack was indistinguishable from that of a convulsive seizure. Apart from these attacks the man was perfectly normal and was fully occupied in useful work.

Occasionally inexplicable head-on collisions between motor cars on the open roads may be accounted for by the momentary "black out" of an attack of petit mal, or by a transitory post-epileptic automatic state. This possibility was graphically brought to my notice recently in the case of a young man who was sent to me suffering from epileptic seizures. A short time before he had crashed a vehicle of a local authority into the front of a house, killing several occupants. According to his own evidence the accident was the result of a momentary amnesia and not to the cause which was found by the Coroner's Court. More will be said on this subject when the question of compensation and litigation is considered.

Electroencephalography.[1]—As stated in Chapter III, oscillations of electrical potential normally occur on the surface of the head at the rate of about ten per second when the eyes are closed and the mind at rest. In normal activities of the brain the Berger rhythm, or alpha waves as they are called, may disappear. Alterations in character of these waves or loss of their rhythmicity is indicative of abnormal activity within the brain, even if this does not manifest itself either as subjective or objective neurological phenomena. Abnormal changes in the electrical waves of the brain always occur during an epileptic seizure. Commonly they are seen just before and occasionally in the interval between fits.

A high-voltage wave is thought to be due to an excessive discharge of a group of neurones and is known as hypersynchrony. Loss of normal rhythm is spoken of as dysrhythmia. According to Gibbs, Gibbs and Lennox,[2] grand mal, petit mal and psychomotor attacks have distinct wave formations. In grand mal the waves increase in frequency up to thirty per second and appear as sharp spikes. In petit mal, rhythm is slower than normal and spiked alternately with rounded or flat-topped waves. In psychomotor attacks the rate is slower than normal and the flat-topped waves predominate.

[1] Russell Brain, W "Recent Advances in Neurology," 4th ed. London, 1940

[2] Gibbs, F. A., Gibbs, E. L., and Lennox, W G "Epilepsy: A Paroxysmal Cerebral Dysrhythmia." *Brain*, 1937, 60, 377.

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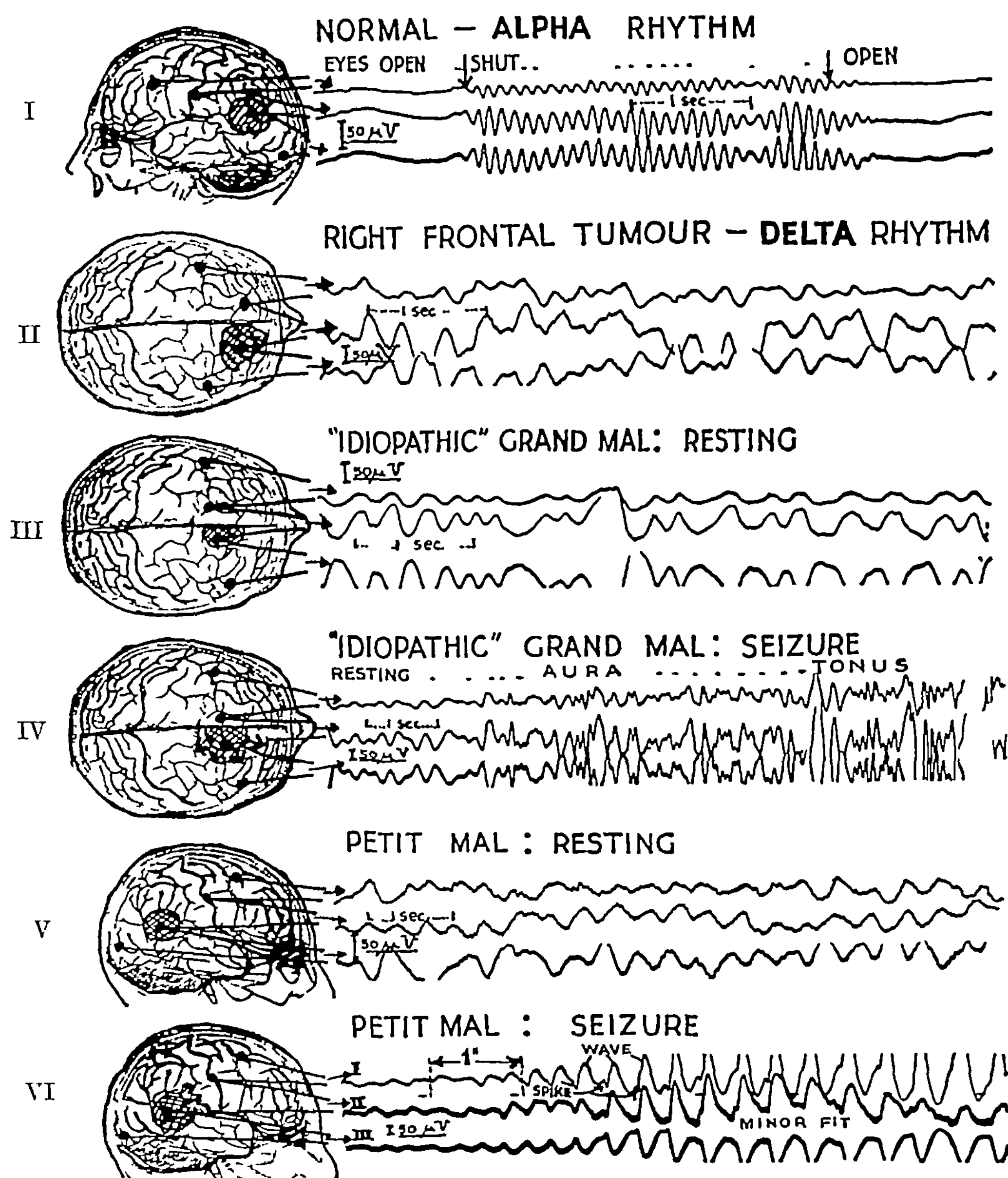


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VI, Seizure rhythm in a patient with petit mal

from corresponding and symmetrical sites on two sides of the brain, or as a diffuse simultaneous discharge.[1]

Delta waves are slow and heaving in character and occur at random frequencies. They indicate that some part of the brain in the region from which they emanate is electrically "dead" and therefore diseased. Thus electroencephalography can not only localise an epileptogenic focus and throw light on the type of seizure that occurs but may also detect and localise a pathological focus (Fig. 119).

Encephalography.[2]—By encephalography is meant radiographic visualisation of the ventricular system, basal cisterns and subarachnoid spaces over the cerebral hemispheres, through the replacement of quantities of cerebrospinal fluid by oxygen or air via the lumbar or cisternal route. In order to determine with precision

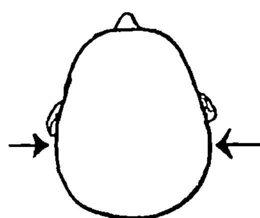


FIG 120
The X-ray position of a "nose-up lateral shoot"

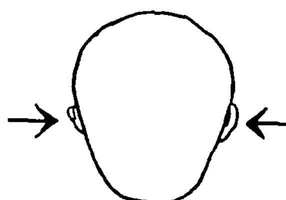


FIG 121
The X-ray position of a "nose-down lateral shoot"

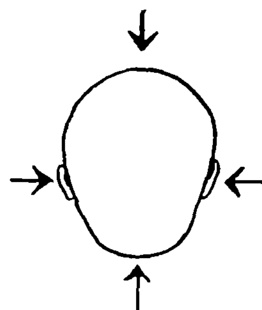


FIG 122
Views taken in two axes

the pathological state of the brain in cases of post-traumatic epilepsy, at least 75 c c of the chosen gas must be injected Moreover, positions other than the routine ones are necessary. For example, if there is a filling defect over the frontal lobe of the brain or in the anterior horn of the lateral ventricle, a "nose-up lateral shoot" (Fig. 120) is useful, as this position ensures that the air must collect in the frontal region if pathological obstructions are not present The "lateral shoot nose-down" (Fig. 121) position is useful when a lesion is suspected or proved to be in the occipital region. It is scarcely necessary to point out that air is lighter than cerebrospinal fluid and collects in the uppermost part of the intracranial cavity Therefore when other special views are needed the head must be so oriented that the suspected area is uppermost The opposite, of course, applies when the skull

[1] Jasper, H , and Kershman, J "Electroencephalographic Classification of the Epilepsies" *Arch Neur and Psych*, 1941, **45**, 903

[2] Shanks, S C, Kersley, P, and Twining, E W "A Textbook of X-ray Diagnosis" Lewis & Co London, 1938-39

is being examined, since a fractured area is shown in greatest detail when placed nearest to the film. Stereoscopic views should be used as a routine procedure, but when this type of examination is not possible, views taken in two axes, with the head in the same position, should be filmed (Fig. 122). With careful technique all gross lesions of the brain and many of lesser degree can be accurately localised and outlined.

The following is a list of the encephalographic findings which I have obtained in cases of post-traumatic epilepsy (Fig. 123) :—

1. Normal ventricular outlines and normal subarachnoid spaces.
2. Normal ventricular outlines with excessive collections of air over the cortex, suggestive of faulty absorption of the cerebrospinal fluid at the arachnoid villi.
3. Normal subarachnoid spaces and bilateral dilatation of the ventricles indicative of obstruction to the circulation of cerebrospinal fluid within, or at the exit of, the ventricles. The obstruction, of course, in these cases could not have been complete, otherwise air would not have entered the ventricles An alternative explanation of the ventricular dilatation is bilateral cortical atrophy.
4. Unilateral dilatation of a lateral ventricle This is indicative of a unilateral cortical atrophy. When displacement of the ventricle is also present, this means that the atrophy is associated with a meningocerebral scar.
5. Porencephaly In these cases a large circumscribed cavity was seen either in communication with the ventricles or with the subarachnoid spaces
6. Local filling defects in the subarachnoid spaces suggestive of meningocerebral scars [1]
7. Flattening of the lateral ventricular outlines with displacement, suggestive of a space-occupying lesion such as abscess, tumour or subdural hæmatoma
8. Filling defects in the body of the lateral ventricles without ventricular displacement, suggestive of intracerebral hæmorrhage.

At the outset it must be realised that encephalography does not and cannot localise an epileptogenic focus which often is distant from a pathological lesion ; neither can it shed any light on the nature, extent or pattern of a seizure. It does, of course, give information on the gross structural state of the brain, and often with much greater precision than any other kind of examination.

[1] Olivecrona, H "Corticomeningeal Scars in Traumatic Epilepsy" *Arch Neur and Psych*, 1941, **45**, 666

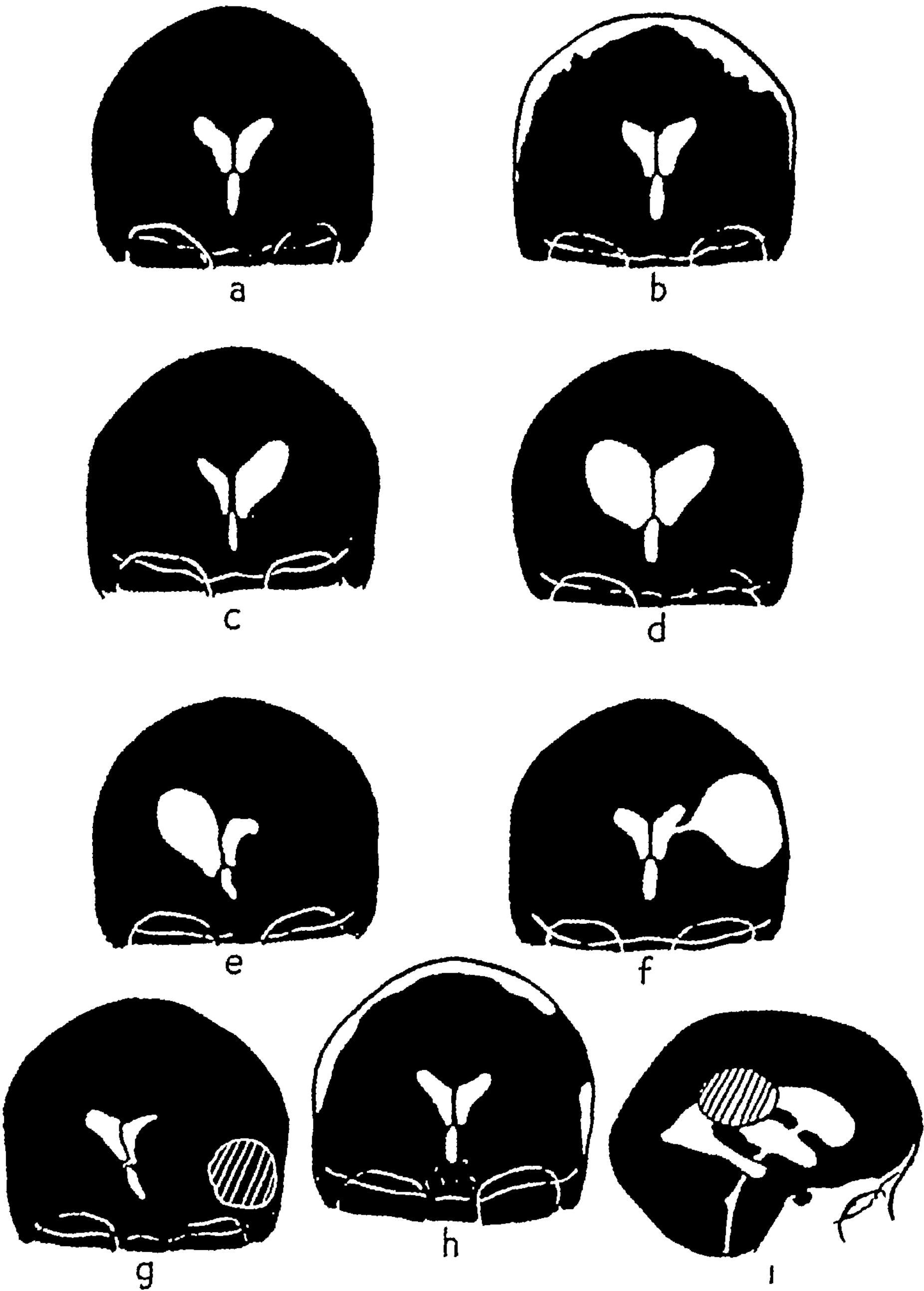


FIG 123

Encephalographic findings in post-traumatic epilepsy

a, Normal ventricular outlines
b, Dilated subarachnoid spaces
c, Unilateral hydrocephalus
d, Bilateral hydrocephalus
e, Unilateral hydrocephalus with displacement
f, Porencephaly
g, Displacement due to abscess
h, Surface-filling defects due to meningocerebral scars
i, Filling defects in the lateral ventricle

TREATMENT

The treatment of post-traumatic epilepsy is largely an economic rather than a medical or surgical problem, particularly as the condition rarely leads to neurosomatic degeneration, in which state a patient becomes mentally impaired and his body deformed by spastic paralysis. Even in severe cases epileptic episodes occupy a very short period of a patient's life and often not more than a few minutes or hours in a year.

Apart, therefore, from a few obviously dangerous occupations, such as car driving, working on scaffoldings or on the edge of vats, a man suffering from occasional epilepsy may live a normal life and must be encouraged to do so. There are dangers, of course, in working amongst machinery, and the best occupation for an epileptic is work on the land. Alternatively, whenever a man is found to be suffering from epilepsy it should be incumbent on the doctor to point out the dangers of driving and to urge him to cease doing so, as my experience in the following case will show. Some years ago a patient of mine received a large sum of money in compensation for epilepsy following an injury to his head. With the proceeds he bought a motor car and during a seizure crashed into a telegraph pole, killing himself and seriously injuring his passengers.

Medical Measures.—A patient suffering from epilepsy may be allowed a normal diet, but should take regular meals so that his blood sugar is never allowed to fall to a low level. When regular feeding is not possible, sweets or barley sugar should be carried and taken on those occasions when something more substantial is not obtainable. Also, sweetened-milk drinks taken before retiring to bed may avoid those epileptic episodes which occur early in the mornings or immediately after getting out of bed. Excessive physical strain, excitement or worry should be avoided. If drugs are supplied they must be given in sufficient quantities to give the desired result, but not in doses which cause toxæmia or drowsiness. A routine which I now adopt is to give $\frac{3}{4}$ gr. epanutin in the morning and $\frac{1}{2}$ gr. luminal at night, these amounts being increased as is found necessary.

Surgical Measures.[1]—For patients whose epileptic episodes are of such severity or frequency in spite of medical measures that their means of livelihood and mode of living are seriously affected, the necessity of surgical treatment will have to be considered. Also, surgery is justified in suitable minor cases when for professional, social or domestic reasons epilepsy, of however

[1] Penfield, W "Epilepsy and Surgical Therapy" *Arch Neur and Psych*, 1936, **36**, 449.

infrequent occurrence or mild a character, will lead to far-reaching consequences.

The objects of surgical treatment are twofold: (1) to remove a pathological lesion which is thought to be the cause of the epilepsy, and (2) the destruction of that focus of the brain in which an epileptic seizure originates.

Indications for Surgical Treatment.—By clinical and electro-



Fig 124

A collection of air in the dimple of a cerebral scar over the frontal lobe

encephalographic means that part of the brain in which the origin of a fit is suspected is identified (Fig. 124). Possibly the two foci localised by these methods do not coincide and a decision has to be made as to which is giving the more reliable information. When an aura develops slowly and permits careful analysis, clinical evidence may be accepted and acted upon. On the other hand, when an aura is rapid and precise analysis is not possible, the findings of electroencephalography must be given preference. A pathological focus is determined either by neurological signs

or by encephalography. Usually a combination of both these methods is necessary. When a pathological lesion coincides with an epileptogenic focus then exploration is indicated without any question. In those cases when a pathological process is distant from the site at which an epileptic seizure starts, it is often difficult to decide whether to operate or not. Providing other methods have been given a fair trial and have failed to give relief, I believe that it is justifiable to explore the brain in order to remove the pathological focus and to diathermise the epileptogenic area if this is also feasible through the same exposure.

Operative Technique.—An abscess is removed together with its capsule; a cyst is drained and, if possible, its wall dissected out; a blood clot is evacuated and drained; a surface aneurysm is ligated and a new growth is excised.

As these conditions are not peculiar to trauma, nothing further will be said here on the operative technique of their treatment. These details may be found elsewhere It is with the excision of cerebral and meningocerebral scars that we are principally concerned.

Exposures are made through osteoplastic flaps, which are so designed that the whole of a pathological lesion together with the epileptogenic focus is uncovered. When for anatomical reasons this would necessitate an excessively large flap, the exposure may be confined to the region of the pathological change. Operations must be carried out under local anæsthesia, drugs, basal or general anæsthetics not being given, as these so damp down the excitability of the cortex that it becomes unresponsive to electrical stimulation.

Let us now suppose that we have exposed the dura mater and that a meningocerebral scar exists. By means of a U-shaped incision the dura mater is reflected upwards. Under the circumstances mentioned this will necessitate division of the adhesions between the cortex and the under surface of the dura As meningocerebral adhesions are tough they do not tear easily, and are best divided by a diathermy current To do this a small metal hook is placed on the far side of an adhesion, and as this is put on the stretch the hook is touched with a diathermy button After the dura has been reflected the exposed cerebral cortex is carefully inspected. From the pattern of the blood vessels and shape of the gyri it is impossible to delineate the motor cortex, and this must be done by means of electrical stimulation with a faradic or galvanic battery, or best, with a thyratron. Care must be taken not to use too strong a current, as a major convulsive seizure may thereby be precipitated from any part of the cortex. This is a very dangerous happening if the veins in the region of the

parasagittal sinus have been exposed, because they may become so engorged with blood as the patient strains that they snap and bleed uncontrollably.

Before going further it must be stressed that no part of the brain subserving a function which is essential to normal life must be injured during any surgical manipulation. For example, it is obviously wrong to leave a patient aphasic or hemiplegic in an endeavour to cure him of epilepsy.

Let us now suppose that the pathological focus may be excised without damage to important areas of the brain. The line of excision, enclosing if possible the epileptogenic focus, is mapped out and the vessels crossing this line are coagulated by a diathermy current. A convenient way of doing this is by the following method. With a pair of fine non-toothed dissecting forceps a hole is made through the pia on each side of the vessel to be coagulated, the vessel then being picked up between the blades of the dissecting forceps and coagulated. Without first making holes in the pia, the toughness of the membrane prevents the vessels from being picked up cleanly. The vessels are then coagulated in like manner half an inch internal to the line of excision and divided with scissors close to the first point of coagulation. Then with a sharp scalpel the incision is deepened and a block of tissue removed as far as the ventricles if the scar extends as deeply as this. The next step is to stop all bleeding points with meticulous care. On those occasions when the epileptogenic focus has not been removed in the block resection of the pathological process, its position ought again to be determined by electrical stimulation and the area diathermised or excised. Personally I depend on diathermy alone for destruction of an epileptogenic focus distant from a pathological process.

The next procedure is to close or repair the dura if tissue has been lost in such a way that adhesions do not reform between it and the cortex. This is best done by covering the damaged area of the brain with a sheet of amnioplastin. Defects in the skull when present should also be repaired at the same operation by one of the methods described in Chapter IV. The slightest degree of infection will prevent healing and nullify any good that might result from the operation, and in fact may leave the patient in a worse condition than before. Finally, owing to lack of tension, post-operative clots are particularly liable to occur in these cases, and therefore before a wound is closed every vessel, however small, must be firmly sealed Anti-convulsive drugs should be given regularly for at least six months following operation, or for as long as may be found necessary.

THE POST-CONCUSSIONAL SYNDROME

In the recovery from any severe injury of the head there are three distinct phases which can be easily recognised and recorded. The first phase is the return of consciousness, which may take any time from a few moments to several weeks, the usual period being a matter of days. Secondly, there is a period of convalescence, lasting about three weeks, at the end of which time the patient is able to get up and about. Following this there is a long process of gradual recovery which may take two or three years before the patient attains his pre-accident state. This third phase is the period of the post-concussional syndrome, and few escape without suffering to a lesser or greater extent from one or a combination of the following conditions: pains in the head, dizziness, insomnia, diplopia, dispositional changes or intellectual impairment.

In the past few years I have examined and taken detailed notes of 500 cases of people suffering from the effects of injury to the head; moreover I have had opportunity of comparing my findings with those of the patients' general practitioners and of other skilled observers. An analysis of the main symptoms of this series of cases will be found in the following table —

ANALYSIS OF SYMPTOMS IN SERIES OF FIVE HUNDRED CASES OF POST-CONCUSSIONAL SYNDROME

Faulty Vision		Deafness	Loss of Smell	Loss of Taste.		Vomiting	Anæsthesia of some Part of the Body	Miscellaneous Symptoms.	Headaches.	
Unilateral Blindness.	General Deterioration of Vision			Partial	Complete				Bilateral.	Unilateral.
8 1·6 %	74 14·8 %	24 4·8 %	27 5·4 %	14 2·8 %	8 1·6 %	14 2·8 %	23 4·6 %	207 41·4 %	295 59 %	102 20·4 %

Dizziness.		Insomnia.	Bad Dreams.	Changes in Disposition	Nervousness.	Unsteady Gait	Insanity		Epilepsy
Labyrinth.	Black-outs.						Temporary	Permanent.	
18 3·6 %	86 17·2 %	116 23·2 %	54 10·8 %	350 70·0 %	315 63·0 %	171 34·2 %	18 3·6 %	15 3·0 %	30 6·0 %

Pains in the Head [1, 2].—Headaches may be unilateral or bilateral; they may come on suddenly and disappear equally suddenly, or they may develop slowly, come to a climax and then recede slowly; they may occur at some particular period of the day; and they may be precipitated or aggravated by exertion, excitement or worry. The most common complaint is

[1] Northfield, D. W. C. "Some Observations on Headache." *Brain*, 1938, **61**, 133.
[2] Schumacher, G. A., and Wolff, H. G. "Experimental Studies on Headache." *Arch. Neur. and Psych.*, 1941, **45**, 199.

of a persistent and generalised discomfort in the head, associated with severe stabs of sharp pain, particularly in those areas where the scalp was bruised or cut. Many terms, such as pressing, boring, lifting, bursting or twisting, are used to describe the type of headache, and this is what might be expected, since the precise description of the qualities of any pain is difficult even for the most skilled observer. Varieties of headache, however, are due not only to peculiarities of individual reception and differences in command of language but also to the ways in which the pain may be produced. Some tissues of the head are sensitive and others are insensitive. Those which are sensitive are the scalp, the periosteum, the meningeal vessels, the dura in the region of the large venous sinuses, and the large vessels at the base of the brain. Those which are insensitive are the bone and diploe as distinct from the periosteum, the dura not in the region of the meningeal vessels or large venous sinuses, and the brain tissue.

Creeping feelings as if insects were moving beneath the skin and other such peculiar sensations or paræsthesiæ are probably the result of contusions or incomplete divisions of the nerves supplying the scalp. In my opinion, scalp pains resulting from traumatic neuritis are commonly the cause of so-called headaches; this diagnosis may be confirmed by injecting a ring of local anæsthetic around the base of the scalp. When testing for relief of pain by anæsthesia a generous amount of anæsthetic must be used and wide areas infiltrated, because there is a considerable overlap from neighbouring nerves in every region supplied by a specific nerve trunk. This etiology of scalp pains is similar to that of causalgia in the limbs, and particularly is this so when the skin has been lacerated and infection has supervened. Unfortunately pathological changes commonly extend along the sheath of the nerves, which do not come to rest until the optic thalamus is reached, when the condition is beyond the redemption of medical or surgical measures

Migrainous types of headache are due to distension of the intracranial and meningeal arteries, and are commonly precipitated by cerebral trauma Typically, the headache starts slowly, increases in severity until what may be called a plateau of intensity is reached, and then the headache slowly recedes. One or, as is usual, both sides of the head may be affected and the pain may radiate into the neck, ears or face. Visual hallucinations, such as black spots or coloured lights, rarely accompany the attack as they do in so-called idiopathic or primary migraine Often when the pain is at its height the patient is completely incapacitated, feels confused and has to lie down or go to bed to get relief. Such headaches tend to have a definite periodicity, coming on in the

morning, afternoon or evening. Though they may last for a few days, they also disappear for similar or longer periods. Often they are brought on by going to the cinema, by excitement or by exertion, and are apt to persist for many years in the nature of recurrent attacks.

Severe and continuous pains are occasionally due to distortions maintained by adhesions causing traction on the sensitive tributary veins draining into the large venous sinuses. Penfield and Norcross [1] were the first to describe this mechanism of pain which is thought to be brought about in the following way. During the moment of violence, whether the skull is distorted or not, the brain slides across the face of the dura at the arachno-dural interface, and for some physical reason, such as hæmorrhage, does not immediately regain its normal position after the violence has ceased to act In this position of subluxation, adhesions form between the outer and inner surfaces of the arachnoid and dura respectively, which later are put on the stretch as the brain tends to take up its normal position in relation to the skull. On those occasions when an adhesion is attached to a sensitive area of the dura—that is, to a large meningeal artery or edge of a dural sinus—pain will be the result.

Pressure or traction on large sensory nerves in the region of the tentorium produces the type of pain that is felt over the forehead or behind the eyes, this projection being explained by the fact that branches of the trigeminal nerve which supply the dura sweep backwards and then upwards and forwards over the vault to end in the frontal region That this type of pain does occur is well demonstrated when performing ventriculography under local anæsthesia by the posterior route. After holes have been bored in the skull and an opening is being made in the dura, the patient will often complain of severe pain at the back of the eyes.

Acute bursting headaches are due to attacks of acute hydrocephalus. These, however, are uncommon, as spinal or ventricular manometry rarely show increased intracranial tension Subnormal cerebrospinal fluid pressures, on the other hand, are much more common and are a frequent cause of headache, particularly when a fracture allows constant leakage of fluid externally or into the subgaleal space.

Finally, headaches of a dull aching kind of indefinite localisation may be caused purely by anxiety or the boredom of unemployment. The conception that neurosis is often the cause of cephalalgia is gaining ground, and this subject will be taken up in greater detail later.

[1] Penfield, W , and Norcross, N C "Subdural Traction and Post-traumatic Headache. Study of Pathology and Therapeusis" *Arch Neur and Psych*, 1936, **36**, 75

Dizziness.—Dizziness, strictly speaking, is a hallucination of movement in oneself, in one's environment or in the third dimension which interferes with, or tends to interfere with, the sense of balance. This type of disorder is relatively rare, and is due to a hæmorrhage into the labyrinth or to damage of the labyrinthine fibres of the auditory nerve. The symptoms are similar to the crises of Ménière's disease, in which the patient is seized with an uncontrollable giddiness and falls heavily to the ground without losing consciousness. In the less severe attacks the ground seems to lift a little, causing a sense of insecurity

Physical or neurological evidence of labyrinthine dysfunction in between attacks is often meagre and in fact may not be demonstrable by simple clinical means, particularly as associated cochlear involvement rarely causes gross impairment of hearing. Before making a final pronouncement on the origin of dizziness, detailed radiographic studies of the petrous bone will have to be made and the integrity of the semicircular canals and cochlea determined by caloric tests and audiometer readings respectively. Fractures of the petrous bone are often incomplete, the upper surface only being involved This means that fracture lines are difficult to demonstrate and will pass unnoticed unless stereoscopic views are taken in the Towne and basal positions. In the absence of radiographic evidence of basal fracture, deafness, facial paralysis or tinnitus point to petromastoid involvement. From the delicate structure of the internal-ear apparatus it will be readily appreciated that dysfunction may occur without gross anatomical damage; thus slight disarrangement may lead to abnormal function. Probably a completely destroyed labyrinth is symptomless, whereas an active but disordered labyrinth will lead to disabling symptoms. The periodicity of attacks may be explained either by variations in the local circulation within the petrous bone, or as is more likely, by certain types of movement of fluid within the canals themselves, causing an unusual stimulus for which the brain is unable to compensate. Hallpike and Cairns [1] explained the periodicity of the dizzy periods of Ménières disease by œdema of the semicircular canals. This condition may be the causative factor in those rarer cases of dizziness when trauma initiates the processes of degenerative otitis interna

Labyrinthine tests consist of stimulation of the semicircular canals by rotation or by the injection of hot or cold streams of fluid into the external auditory meatus. By these means a sense of giddiness and nystagmus of variable types and measurable degree are produced When one ear only has been damaged, the

[1] Hallpike, C S, and Cairns, H "Observations on Pathology of Ménières Syndrome" *Proc Roy Soc Med*, 1938, **31**, 1317, also *Jour Laryng and Otol*, 1938, **53**, 625

labyrinthine responses may be compared with those of the good ear. On the other hand, when both ears are affected the responses of a normal ear must be used for comparison. This is where the co-operation of a skilled and experienced otologist is required if correct deductions are to be made. In fact no case of persistent dizziness can be regarded as thoroughly investigated until an otologist has given his opinion on the intactness or otherwise of the internal ear. Often the audiometer which produces a pure note without overtones will detect a defect in the normal range of hearing which remains undiscovered in conversation or by the watch and tuning-fork methods. Other lesions such as cerebello-pontine angle and cerebellar tumours or otitic meningitis are sometimes present and are apt to be overlooked because of the history of trauma It is therefore wise to approach all cases of dizziness with an open mind and to carry out a carefully planned neurological examination In acoustic neuromas, giddiness and tinnitus are occasionally the prominent symptoms These, however, are usually associated with some degree of deafness, facial weakness, trigeminal hypo-æsthesia as shown by a diminished corneal reflex on the affected side, papillœdema and increase of proteins in the cerebrospinal fluid Thrombosis of the posterior inferior cerebellar artery is often heralded with the most violent attack of giddiness, but this diagnosis is usually a simple matter, since this condition not only produces difficulty in swallowing but also sensory changes on the whole of one side of the body or on the face of the same side and in the limbs and trunk of the opposite side Linthicum and Rand [1] are convinced that labyrinthine changes are commonly demonstrable following severe concussion, and much has been written on this subject by the French school of neurologists.[2] [3]

Usually what the patient means by giddiness is that things go black before his eyes whenever he makes a sudden movement, such as stooping. From the table on page 241 it will be seen that this sensation is far more common than actual hallucination of movement Upward movements of the eyes are particularly liable to precipitate an attack. These momentary black-outs probably are due to instability of the cerebral circulation, consequent upon injury to its vasomotor apparatus rather than to labyrinthine dysfunction Thus the essential cause of most giddy attacks is momentary ischæmia of the brain consequent on circulatory lability

Ataxia and loss of balance due to gross lesions of the cerebellum

[1] Linthicum, F H, and Rand, C W "Neuro-otological Observations in Concussion of the Brain" *Arch Otolaryng*, 1931, **13**, 785

[2] Barré, J A *Rev d'Oto-neuro-opht*, 1932, **10**, 385

[3] Portman, G, and Delmas-Marsalet, P *Rev d'Oto-neuro-opht*, 1933, **11**, 23

or its connecting pathways are exceedingly rare. In any case these dysfunctions are chiefly objective rather than subjective in nature. From the prognostic point of view accurate diagnosis is essential since circulatory disturbances usually recover spontaneously, whereas physical injuries to the labyrinth often necessitate surgical intervention, such as division of the labyrinthine fibres of the eighth nerve or complete destruction of the labyrinth with alcohol before relief can be obtained.

Insomnia.—Insomnia is a most troublesome symptom not only in itself but inasmuch as it gives rise to a vicious circle. So long as it exists, recovery of general health is improbable, yet on the other hand insomnia is unlikely to improve so long as the patient feels mentally and physically ill. Whether insomnia is the result of structural changes within the brain or to anxiety combined with a general deterioration in health is not known. Probably it is due purely to psychical processes interfering with the normal physiological integrations on which sleep depends. Usually a patient cannot get off to sleep till the early hours of the morning, and when he does he soon wakens again to start the same process once more, with the result that he wakens in the morning feeling more fatigued than when he went to bed. Often he will get up in the night and make himself a cup of tea. Nightmares in which the details of the accident are reproduced are infrequent, although disturbing dreams in which something sinister happens are fairly common. No doubt the rarity of reproduction or imaginary repetition of the details of accident is linked up with the state of retrograde amnesia, that is, blankness of memory for events immediately preceding unconsciousness. In people over sixty, especially those with high blood pressures, even minor injuries may result in intractable insomnia

Diplopia.—In the acute stages of severe concussion, when a patient is still unconscious, skew deviation and inco-ordinate wandering of the eyes are often seen. Probably these are due to disturbances of the ocular nuclei within the brain stem, caused by petechial hæmorrhages or faulty circulation in the radicles of the basilar and cerebellar arteries. Most cases of double vision are the result of the continuation of this state, although in a lesser degree. The fault in the affected muscle is often so slight that it is difficult to prove which muscle is causing the diplopia. Recovery in this type of case is spontaneous and rapid.

Gross paralyses of extraocular muscles, causing an easily discernible squint, are usually due to injury to the trunks of the third, fourth or sixth nerves themselves as they enter or traverse the orbit. Rotations of the brain stem occasionally lead to attrition of the sixth nerves as they lie within the posterior fossa.

Recovery in these cases is doubtful. The prognosis is favourable when improvement starts early or when the paralysis is partial, but it is doubtful if no improvement has occurred within three months.

Fractures frequently involve the orbit, causing deformity of the orbital cavity with resulting displacement of the eye. This happening fortunately is not necessarily accompanied by diplopia, because the brain is often able to compensate for the displaced eye by fusing the images which come from it with those from the other eye, or by suppressing them altogether. Compensation, however, rarely occurs when neuromuscular mechanisms are permanently affected

Changes in Disposition.—The anatomical and physiological basis of emotion is thought by some to be seated in the thalamus and other centrally placed nuclei. Outbursts of rage occur in patients with tumours affecting the hypothalamus, and surges of uncontrollable temper and homicidal tendencies have been observed in cases of disease of the thalamus. Similar states occasionally follow head injuries, possibly due to small hæmorrhages in the thalamus and hypothalamus

The usual clinical picture, however, is not one of violent extremes It is that of a previously good-tempered happily minded man becoming irritable, intolerant, selfish, disinterested and miserable. Instead of being concerned in the well-being of his family he will refuse to work, and by his behaviour will focus the whole attention of his wife, relations and friends on himself He loses interest in the things which used to engage his attention and will sit for hours gazing into the fire without troubling to answer when spoken to He finds noise of any kind irksome, he is peevish and avoids social contacts Self-confidence and self-reliance completely disappear. There are, of course, changes of a lesser degree which are obvious to intimate relatives only.

Of all the symptoms of the post-concussional syndrome, changes in disposition are the most distressing to the family concerned, particularly as complete recovery is often not made. The underlying pathology of this condition is probably the result of widespread physical damage in the brain of a diffuse contusional nature.

Intellectual Changes.—The link between the brain and the mind is not known, but since intellectual processes are dependent to some extent on the experiences of the special senses which are represented widely over the brain, it is reasonable to presume that the whole brain rather than parts of it are concerned with the intellect. Possibly the frontal lobes are concerned only with the highest intellectual activities

Gross impairment of intelligence is uncommon in the post-concussional state. When it does happen it is due to laceration of the brain, or in other words to gross loss of cerebral tissue.

The usual picture is that of mental sluggishness—an unwillingness to think rather than a total inability to do so. A patient will answer questions slowly but correctly even if this is done in a roundabout way, and he will reason and converse in a perfectly logical manner. In fact his theorising on the cause of his symptoms is often illuminating. He is accessible and can always be made to understand, though not necessarily convinced. Improvement rapidly takes place.

In children of school age intellectual changes are common and most serious.[1] At the very least the child's educational training is retarded by the length of the illness, which is often the equivalent of one year, for even though the child returns to school in a few months his new studies are incomprehensible to him without the earlier grounding leading up to them. The child gets behind, is thrown with strange companions and becomes discouraged. In any case, if he is not kept at school beyond the usual leaving age for a period equivalent to that which he has lost, his final educational standard must be lower than it would otherwise have been. The parents of children of the upper and middle classes are well aware of this fact and act accordingly, but unfortunately the children of the working classes are usually not safeguarded in this respect as they should be

Before school age the effects of injuries on the intelligence cannot be assessed save in those cases where they cause frank mental deficiency

Neurosis [2].—A neurosis is essentially a disorder of the personality not based on physical injury within the brain of any particular pattern On the other hand it is often the result of the reaction of the mind to objective or subjective effects of a structural pathological state in the brain Every neurosis, however, following an injury to the head does not necessarily imply a physical injury to the brain, as the state may be produced purely by psychological trauma or the mental shock of the accident Prolonged trauma, such as occurs in industrial accidents when a man is trapped and crushed and does not lose consciousness immediately, is almost invariably followed by a functional overlay with the attendant train of symptoms of which loss of confidence and tremor are the predominant features.

[1] Blau, A "Mental Changes following Head Trauma in Children" *Arch Neur and Psych*, 1936, **35**, 723

[2] Schilder, P "Neuroses following Head and Brain Injuries" 'Brock's Injuries of the Skull, Brain and Spinal Cord" Baillière, Tindall & Cox London, 1940

Purely neurotic syndromes following minor injuries are common, and the prognosis in these cases is capricious, since the breakdown may be due mainly to business worries, domestic unhappiness or frustration of ambition, and these conditions must be corrected before improvement can be expected.

Psychoses [1].—These may be primary or secondary. In the first case the mental disorder is due directly to a physical injury of the brain, whereas in the second case a pre-existent degenerative process or latent psychotic state is merely precipitated by the injury. Typically the sequence of events in a primary psychosis is as follows. A patient is rendered unconscious by a blow on the head Instead of approaching consciousness through the ordinary phases of irritability he becomes raving mad and so uncontrollably dangerous to himself and those around him that he has to be transferred to a mental institution where better facilities obtain for the treatment of this kind of patient As the man becomes more conscious it will be seen that he is grossly disoriented regarding time and place and is quite oblivious of his circumstances, although he may evidence distress regarding physical discomforts. Usually with time and rather rapidly he regains insight, becomes biddable and very soon is normal as far as his psychotic state is concerned. Only occasionally does the mental state retrogress, the man becoming an obvious lunatic In this state his physical condition also deteriorates He becomes incontinent, his skin sags, he loses weight, also his circulation becomes sluggish, as shown by cyanosed extremities and a blotchy complexion. The prognosis of a primary psychosis in the absence of a history of mental instability is good

Secondary psychosis may be subdivided into two groups. The first group embraces those cases which fit into a formal classification of dementia præcox, paranoia and mania, etc. These are merely precipitated by the injury, as shown by the fact that they may follow trivial injuries to the head which could not have produced cerebral damage sufficient in degree or extent to account for them

In the second group a degeneration of the nervous elements takes place after an interval of a few weeks or even years, which proceeds far beyond the limits of the original injury, leading to profound mental defects which declare themselves clinically in a variety of ways. A good example of this secondary change is a condition known as "punch drunk," seen at the end of a long career in boxing Repeated jolts to the head cause petechial hæmorrhages into the brain substance which at the time pass

[1] Symonds, C P "Mental Disorder following Head Injury" *Proc Roy Soc Med*, 1937, **30**, 1081

unnoticed but which may later lead to mental impairment and loss of vision.

In arteriosclerotics, syphilitics and alcoholics, trauma to the head is particularly liable to initiate a diffuse and degenerative process in the brain tissue. Minor degrees of violence that would cause no serious structural damage in a healthy brain may, under the foregoing conditions, lead to progressive insanity. In the primary and formal psychotic groups accompanying neurological signs of damage to specific parts of the brain are sparse or absent, whereas they are marked in the secondary degenerative type of case. Prognosis in a primary traumatic psychosis is good, poor in a formal psychosis and hopeless when a degenerative encephalopathy is present.

THE EXAMINATION

Let us start at the moment when a man, complaining of the results of an injury to his head and with litigation at stake, walks into the consulting room. Usually he enters slowly and hesitantly, accompanied by his wife and often with other relatives or friends. He will stand about aimlessly until told what to do and will rarely attempt to take off his overcoat or get rid of his hat until his wife does it for him ; she also sees him safely back to his seat Characteristically he will sit perfectly still staring straight in front of him with a fixed expression of peculiar indifference on his face It is the demeanour of his wife that shows how profoundly unhappy they both are When invited to tell his story he rarely speaks spontaneously and says all he wants to say in a very few words When asked a question he will almost invariably wander round the point and even when pressed rarely gives a straightforward answer Incessantly he will appeal to his wife to answer his questions for him ; many times she will do this without invitation or against instructions not to do so Rarely from the man alone is it possible to get reliable details of his ailments or of his convalescence. Often he will omit to mention one or more of his more prominent symptoms ; also, symptoms are described in the vaguest language. His attitude to the doctor appears to be one of . " You don't understand nor does anyone else, and I can't tell you ; and if I could it wouldn't do any good." His wife, though rarely voluble, gives her opinion in a very earnest manner, being obviously gravely concerned about the whole affair Finally the man usually complains of pains in his head, giddiness and insomnia or occasionally of something specific, such as deafness and loss of taste It is, however, from the wife that the true state of affairs is ascertained. From her it will be understood that her husband is a changed

man both mentally and physically. He has lost his initiative and his interest in everything and everybody. He will sit for long periods staring in front of him without saying anything to anybody. He is unfriendly and avoids social contacts. He is irritable, loathes noise, objects to the wireless and is peevish with the children From his wife he expects and demands the minutest attention. In fact he becomes a profound valetudinarian. He is unable to concentrate; for example, he will read a few words of the paper and then put it away. Memory for recent and immediate events is faulty. Typically he will go into the next room to get something and return without it, having forgotten what he went for and apparently quite oblivious of the fact that he did go for something If sent on a message, instructions have to be written down or he would not remember what they were. He sleeps badly; for long periods he remains awake and often gets up to make himself a cup of tea. Later he returns to bed and finally does not get up until noon or thereabouts to start his life of lack of purpose once again. Usually his appetite is not impaired. His general health looks excellent and he is often tanned in comparison with his pale-faced wife and fellow-workmen. Sex relationships radically change, a passionate attentive man will become indifferent to his wife and often brutal in his behaviour.

Volubility on the part of the patient when taking a history is rare, but emotional breakdowns, tears and despairing gestures are common.

After the taking of the history there starts the fuss of getting ready for the examination. Speed of undressing in my experience is a social distinction A working man always does this slowly and often loses his stud, whereas a professional man undresses rapidly. When complaining of the post-concussional syndrome, any patient takes an unconscionably long time to take off his clothes and has to be helped by his wife. As a matter of interest I once allowed a man to undress and dress at his own speed and timed him. He took exactly twenty minutes to undress and half an hour to dress. If spoken to when about to untie his bootlace a patient will stop all movements, sit upright, think about what he is going to say and then say it slowly. When undressed he will complain of the cold and his wife will give you the impression that you are doing him serious harm by stripping him. It will be found that his gait is slow, deliberate and a little unsteady when on the turn. Bending and rotation tests are carried out slowly, clumsily and gingerly. It is, however, rare to precipitate a true attack of vertigo by movement, and although a patient may stumble a little he rarely falls or lurches badly. Looking up may cause him to bend

his head and put his hands over his eyes. Nystagmus of an abnormal type is rarely seen. Usually co-ordination of movement is grotesque. There is often a gross ataxia when attempting to touch the nose with the finger or the knee with the opposite heel, also, the patient may tremble all over his body when asked to do things a little more expeditiously.

Occasionally a patient will roll and lurch about the room knocking everything down but himself, or may shiver so violently that things in the room vibrate On attempting to manipulate the legs they are held voluntarily rigid. The knee jerks may be hyperactive or obliterated by voluntary spasm The plantar responses are almost invariably flexor even when the knee jerks are wildly exaggerated Ankle clonus is usually absent But what is interesting is that the abdominal jerks are often absent or inactive. Frank paralysis of the limbs or areas of true anæsthesia about the body are exceedingly rare. The heart rate is fast but varies according to the stage of the examination at which it is taken. At first it may be racing, but later, as the patient's confidence is gained, it often settles down to normal, save in those cases where a general tremor persists. Except in conditions of frank poverty a man rarely loses weight because of his illness When he does he looks very ill Menstruation in young women often becomes irregular or disappears for a time In the several pregnant women who have been under my care, accident has not brought on parturition. The optic discs rarely show any change The fields of vision are reputed to be constricted, but apart from patterned defects I have been able to place little reliance on charting of the visual fields because of the patient's stupidity, unwillingness or inability to co-operate Blood pressure in the absence of circulatory disease is rarely raised.

The above description is, of course, of a prototype embracing the worst features of the post-concussional syndrome At the other end of the scale is the soldier who, without exaggeration of word or deed, declares that he has headaches or suffers from dizziness or other such symptoms.

Apart from destruction of localised parts of the brain or special organs producing such obvious conditions as hemiplegia, aphasia or deafness, the summary of the neurological evidence in the majority of cases is mental and physical sluggishness and dispositional changes It will be realised, therefore, that in the absence of localising signs further opportunities for observation and investigation will be necessary before a final opinion can be made on the genuineness of the symptoms and what the nature of the underlying pathological change in the brain might be

SPECIAL INVESTIGATIONS

Radiography.—Though a fractured skull in itself is of no very great importance, it does point to the fact that the head has been subjected to a considerable amount of trauma Moreover, radiography may reveal injuries to specialised structures such as the middle and internal ear or the optic canals which not only confirm that certain symptoms are genuine but also explain the mechanism by which they were brought about.

Lumbar Puncture, Spinal Manometry and Encephalography.—At the time of lumbar puncture the pressure of the cerebrospinal fluid should be measured so that symptoms such as headaches, the result of increased or decreased intracranial tension, may be eliminated or confirmed. In sixty cases suffering from the post-concussional syndrome the results of spinal manometry were as follows . in one the pressure was high ; in thirty the pressure was normal ; and in twenty-nine the pressure was in the low normal register, normal pressure in the lateral position being regarded as lying between 50 and 150 mm. of cerebrospinal fluid. Low pressures were most commonly found following cerebrospinal fluid leaks and in debilitated patients of poor nutrition and weak circulations. High pressures are only found in those cases with frank pathological changes in their brains such as hydrocephalus and subdural hæmatomata or hygromata. In all cases, cytological and chemical examination should be made of the cerebrospinal fluid, since abnormal findings may lead to the discovery of an unsuspected lesion such as a neoplasm.

The Queckenstedt Test.—On bilateral compression of the jugular veins the column of fluid in the manometer should rise at least 20 cm in ten seconds and fall to the original level within fifteen seconds. This is known as the Queckenstedt test. Any deviation from normal in the way of a diminished rise, of a slow or of a stepped fall is indicative of obstruction within the cerebrospinal fluid pathways ; this usually is in the nature of meningeal adhesions. The importance of demonstrating the presence of adhesions is that they may be the cause of meningeal pain or may produce attacks of so-called acute or transient hydrocephalus with headaches of the bursting type.

Encephalography.—When localising neurological signs are present or intractable pain or other persistent symptoms are complained of, an encephalographic examination should always be carried out, otherwise gross pathological lesions not detectable by clinical methods may pass unnoticed. In particular, thin surface clots or subdural collections of fluids may be demonstrated, the drainage of which will lead to cessation of symptoms. Also,

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supply the necessary information. In my opinion an injury which leads to unconsciousness of more than an hour's duration is always associated with a diffuse neuronal injury of a structural type in which many cells are permanently destroyed. A man who receives such an injury is not able to sit up immediately; moreover, he is more than confused when admitted to hospital and does not speak coherently for many days. Unconsciousness which develops after a latent interval is not due to intrinsic damage, and therefore is not so liable to be associated with permanent structural changes within the brain. With a knowledge of the type of man we are dealing with and of the nature of the injury he received we are better able to understand the effects of the injury.

For example, when it is known that a man complaining of aches and pains was well balanced mentally before his accident and has received an injury to his brain of the structural type, then it is reasonable to presume that some of his symptoms at least have an organic basis On the other hand, functional influences must account for symptoms following minor injuries, particularly when they occur in a man of pre-accident neurotic tendencies. An organic basis may also account for vague symptoms in those cases where there is frank evidence of local brain damage, such as hemiplegia, hemi-anæsthesia, aphasia or loss of smell

Whether headaches are due chiefly to neurotic or organic influences is almost an insoluble problem, for the good reason that they are so essentially subjective in nature. As far as I can see, when there is an element of real pain in contradistinction to an ache, they are usually of organic origin. Evidence of meningeal adhesions or hydrocephalus, of course, confirms this view. On the other hand, dull aching pains of an indefinite character unrelated to the time of day, climate,[1] excitement or exertion are often neurotic in origin.

Dizziness is almost always due either to instability of the cerebral circulation or to contusion of the labyrinth or eighth nerve.

Apart from frank intellectual impairments due to bilateral frontal lobe contusions or to degenerative encephalopathies, it is difficult to attribute faulty memory and slowness of the mental processes to anything but psychological trauma. Alternatively, it must be admitted that the intellect is probably represented throughout the brain and therefore may be affected in cases of widespread neuronal destruction.

Of particular interest are those subtle dispositional changes which are noticeable only to close relations. Often they are so

[1] Mitchell, S W 'The Relations of Pain to Weather being a Study of the Natural History of a Case of Traumatic Neuralgia" *Amer Jour Med Sc*, 1877, **73**, 305

a man's general intellectual and locomotor poverty may be shown to be attributable to a diffuse loss or atrophy of cerebral tissue, as shown by enlargement of the ventricles, basal cisterns and gyral spaces

General Metabolic Overhaul.—The danger of specialism is the tendency to attribute every symptom to the nervous system, forgetting that other organs of the bodily economy may be at fault. Therefore a full examination should be made of the respiratory, circulatory and urinary systems and of the blood, including cell counts and the Wasserman test Cases of Bright's disease, malignant circulatory hypertension, syphilitic arterio-sclerosis, diabetes mellitus and other disorders may easily be overlooked if their possible presence is not sought for by the usual simple methods. As a final word on the subject, the urine should be examined and the blood pressure taken as a routine measure.

Psychological Overhaul.—Whenever a frank and troublesome functional element is present the help of an experienced psychiatrist should be sought and the patient passed over early to him for treatment, if it is the opinion of the doctor that medical and surgical measures associated with time and rehabilitation will not effect a cure

DIAGNOSIS AND ASSESSMENT OF SYMPTOMS

When all the clinical and investigational data have been collected the next problem to be solved is whether the symptoms complained of are genuine, and if they are, what the underlying pathological state which causes them may be.

First it is important to get a clear picture of the man's pre-accident background. Was there evidence of a neurotic tendency? Was he a good husband? Was he a good workman? Usually this information is given faithfully by his wife or relatives, confirmation easily being obtained by consulting his employer Poor types commonly have a characteristic stamp about them, but it is very wrong to believe, as do so many clinicians, that it is possible to weigh up a man's character in a few minutes Such beliefs often lead to grave injustices of judgment Also, it must be realised that an ill-educated working man is at a great disadvantage when attempting to state his case, being apt to irritate the examiner unduly.

The next point that must be determined is the nature and severity of the original illness. In particular, did the man receive a structural cerebral injury and of what degree? In closed head injuries this has to be judged by the length of time he was unconscious In compound injuries the surgeon's notes will usually

supply the necessary information. In my opinion an injury which leads to unconsciousness of more than an hour's duration is always associated with a diffuse neuronal injury of a structural type in which many cells are permanently destroyed. A man who receives such an injury is not able to sit up immediately; moreover, he is more than confused when admitted to hospital and does not speak coherently for many days. Unconsciousness which develops after a latent interval is not due to intrinsic damage, and therefore is not so liable to be associated with permanent structural changes within the brain With a knowledge of the type of man we are dealing with and of the nature of the injury he received we are better able to understand the effects of the injury.

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slight that it is unlikely that they can be due to contusion of the thalamus or to the centres of rage which are situated in the central basal ganglia Diffuse degenerative lesions of the cerebrum of vascular origin are commonly associated with emotional breakdowns extremely distressing to the observer. By analogy it might, with reason, be presumed that like symptoms may be caused by the diffuse lesions of trauma Also, emotional changes are often most pronounced in cases of aphasia, which suggests that emotion may have the same representation in the cortex as speech. Alternatively it may be argued that dysphasia more than any other disability is apt to lead to emotional breakdowns.

Retrograde amnesia, in my experience, has only occurred in those patients who were rendered unconscious immediately on the receipt of trauma and who remained in this state for more than an hour or so. In other words, it occurred in those patients who had received the type of accident that causes structural neuronal damage and not in those who merely received a severe mental shock or a neuronal injury of physiological dimensions When it does occur it is permanent, and the memory for past events never returns. Often a patient will reconstruct what has happened from knowledge that comes to him later, believing that his memory has returned, but it is wise never to accept such evidence as reliable. The events occurring at the time of accident never enter the memory.

Grotesque co-ordinations of movement so commonly seen in the post-concussional syndrome cannot be ascribed to any known kind of injury or to any neurophysiological mechanism which is capable of producing such locomotor disorders Presumably, therefore, they are functional in origin The same argument also applies to shaking and trembling of the whole body. Fine tremors, the result of post-traumatic thyrotoxicosis, sometimes occur. In these cases the diagnosis is usually obvious because the patient loses weight, the pulse is fast, sugar appears in the urine and exophthalmos may develop On the other hand, post-concussional functional states rarely lead to loss of weight or to other signs of disordered metabolism

Progressive degenerative encephalopathies rarely give rise to difficulties in diagnosis, as they are usually accompanied by frank and diffuse neurological signs, such as increasing spasticity, mental deterioration, deafness and optic atrophy.

Although in the present state of our knowledge any analysis of what is functional and what is organic can only be done with some misgiving, an attempt must be made in order to establish the premises of the final argument. It must be realised, however, that the difficulties do not end here, for even if the above data

could be assembled with accuracy the problem of diagnosis would still remain complex, because the patient's final state cannot be the sum of the separate influences of the organic and functional but must result from a complicated integration of both. For example, the abnormal and unpleasant sensations arising from circulatory influences must affect adversely any neurotic state which may result purely from the mental shock of the accident and tend to perpetuate it. Alternatively, the unpleasant element of sensations resulting from contusions, etc., will be intensified by a neurotic receptive state.

It is well to realise that complexities do exist and to face up to them from the outset. Atypical syndromes which are difficult to understand are apt to be dubbed psychogenic for the good reason that no other explanation can be thought of. Such deductions, of course, are unsound For example, what is it that accounts for a patient's willingness, after a severe head injury, to live on an inferior level socially and economically? Essentially it is a state of mind that allows him to do so, but such a way of thinking may possibly result directly from a complex physical cerebral state As Straus and Savitsky[1] have pointed out, it is interesting to read accounts by physicians of their own symptoms following injuries to their heads.[2,3] Such people, though originally firm believers in the psychogenic influences in producing symptoms, become converted to the belief that organic changes are really the cause of the post-concussional syndrome. The great argument of the psychogenic theorists is that a patient suffering from the effects of a severe head injury can be rapidly improved by adequate or liberal compensation. The answer to this is, in fact, quite a simple one. Compensation allows a man to readjust himself more easily than one who has not been compensated, and relieves him of family and economic worries at a time of stress. Surely a man's mental state must deteriorate if, because of his illness, his family becomes impoverished and has to accept charity to subsist. Recently I have had the opportunity to go into the homes and talk with the relatives of people injured many years ago There the results of accident can be seen in their true perspective. The opinion of the injured themselves and their families is always the same. Even when generously compensated, the injuries have always had far-reaching and sad results. The husband has lost his position in the home, his wife has had to

[1] Straus, I, and Savitsky, N "Head Injury Neurologic and Psychiatric Aspects" *Arch Neur and Psych*, 1934, **31**, 893.
[2] Durand-Weaver, M "Die Commotio Cerebri und ihre Bewertung" *München Med Wchnschr*, 1929, **76**, 1879
[3] Mayer "Die Commotio Cerebri und ihre Bewertung" *Munchen Med Wchnschr*, 1929, **76**, 2135

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being dependent on actual disarrangement or dissociation of the neural mechanism concerned, which explains why fixed postures can be maintained without fatigue. Moreover, the mental attitude of the patient is such as to give the impression that he is pleased with his disability and courts rather than avoids interest on the part of the examiner.

The Detection of Malingering.—The true malingerer often comes under suspicion at the first interview because of obvious lack of frankness and sly looks. Unlike the genuine case, he takes an intense interest in what is going on around him, although he tries hard to disguise this fact. When asked a question he thinks out carefully what he is going to say before giving his answer, which is often in the form of a reasoned argument rather than of the more usual careless and imperfect reply

Also, there is a marked willingness for him to theorise on the cause of his illness in a way that is not met with in more genuine cases. During his physical examination he is slow to co-operate rather than awkward or inattentive. Also, he tends to exhibit his feelings by grotesque contortions of his face, particularly when he claims that he is being hurt. Occasionally his attempts at stooping or bending will be ridiculous When pretending to move a painful or stiff joint the antagonist as well as the agonist group of muscles will go into spasm Sometimes he makes the mistake of complaining of some specific disability, such as paralysis, anæsthesia, deafness, diplopia or blindness, the simulation of which is easy to detect. In these cases, if he does not trip up when his attention is engaged on some other subject he is almost sure to be caught out when special tests are applied. The number of ingenious tests that have been designed for detection of the malingerer is legion. A few only will be discussed here

Paralyses.—These do not conform to any known type and are not accompanied by characteristic reflex changes. When, for example, it is claimed that the leg is weak, the knee jerks are often exaggerated, but the Babinski response is flexor. If the patient knows to point the big toe upwards when the sole of the foot is stroked, the other toes fail to fan or spread outwards. Perhaps the most reliable evidence of simulation of spastic paralysis is the manner the muscles react to stretch. On attempting to flex or extend a joint, the movements of which are restricted by spasticity of pathological origin, the muscles will slowly then suddenly relax, so that the resulting movement is jerky. In cases of malingering, the greater the force applied the greater is the resistance put up by the patient; as the force is

shoulder the burden, and both would give as much as they have received if the accident need not have happened. To try and put these simple statements into more scientific language would destroy their genuineness.

In the ability of readjustment, the personal element and background is of course a dominant factor. There is the thrustful ambitious young man who, just beginning to make a success of his career, feels that he must get back to work even though this be done at the price of considerable physical discomfort. Characteristically, a young athlete by the urge of the game will patch himself up to turn out to play at the risk of doing himself serious harm. It is doubtful, on the other hand, whether with the same physical disability he would be willing or able to stand up to the stress and strain of a long route march for which he had no enthusiasm. It is true that it is a very serious injury which completely incapacitates a man, and such injuries are rare. For example, a professional man will carry out a long day's work in spite of a severe attack of migraine. On the other hand, it would be unnatural for a man to readjust himself in such a way if his hurts were due to other people's carelessness and if his chances in life were such that ambition were virtually non-existent.

MALINGERING

By malingering[1] is meant the deliberate simulation of symptoms and signs for material gain. Such behaviour is, of course, not only reprehensible but unlawful and subject to heavy punishment. Fortunately it is rare. Exaggeration, on the other hand, is common, and in my opinion justifiable in the large majority of cases. When there is an obvious physical disability it is unnecessary, but how else can a man whose ailments are entirely subjective declare to those around him how he feels other than by persistent complaining and by demeanour and attitudinisation. Hysteria, in common parlance, denotes purely an emotional outburst and must not be confused with rare and ill-understood hysterical phenomena. Following physical trauma, hysterical disorders almost invariably occur in the form of an inhibition not as an over-action of function. A paralysis or a fixed deformity of a limb is the commonest form of the disorder, although it may assume any guise; cases of hysterical anæsthesia, blindness, aphasia and sneezing being included in my records. It is not regarded as a true form of malingering, because it is believed that it is not entirely under the control of the volition,

[1] Collie, J. "Malingering and Feigning Sickness." *Longmans, Green & Co.* New York, 1917.

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increased he brings other muscle groups into play to give him purchase.

Blindness.—Following closed head injuries, total blindness, whether genuine or otherwise, is exceedingly rare, and in the many thousands of records to which I have had access it occurred only on two occasions. Alternatively, general deterioration of vision is a common complaint. Usually this is genuine, for though it may obviously be due to non-traumatic conditions such as refractive errors, it is introspection consequent on ill-health that leads to the discovery of hitherto unsuspected physical imperfections In such cases a patient should not be stamped as a malingerer; rather his complaint should be reviewed in the above perspective.

It is the complaint of complete unilateral blindness of which the examiner should be chary. Occasionally the amblyopia of a squinting eye will pass unnoticed until trauma calls attention to its true state, and the patient may claim that his blindness resulted from the accident. Usually the true diagnosis is a simple matter; a gross refractive error is present and there is a history of long-standing squint. In my experience unilateral blindness has never been associated with strabismus and there has always been ophthalmic evidence of optic atrophy. Absence of pathological retinal change is suggestive of malingering To confirm the genuineness of unilateral blindness the following test will be found useful. With both eyes open the patient is asked to read small print at reading distance. When he is doing this a glass prism is placed before the supposedly blind eye. In cases of malingering the resulting distortion of vision will interfere with the sight in the good eye so that reading becomes difficult, whereas in the genuine case no such interference occurs Simulated visual field defects may be exposed by chartings made at different distances from the test object, the area of the visual field varying with the distance at which the test is made.

Deafness.—In bilateral deafness there is usually a characteristic nasal intonation of the voice and an attentive look about the face which is missing in the malingerer. When testing it must be remembered that people who are virtually deaf may be startled by loud noises, therefore the use of a sudden explosion is not a valid test for the detection of deafness. Before deciding on the genuineness or otherwise of a case, wax should be removed from the external auditory meatus and the drum examined If this is not done the examiner will occasionally suffer the annoying experience of someone else making a more accurate diagnosis than he, and effecting a cure by simple means In bilateral deafness the examiner has to depend on the patient forgetting he is

deaf at all times to the spoken word. For example, he may obey a command spoken with the ordinary voice when his attention is otherwise engaged. Another simple test is ostentatiously to plug the sound ear with a ring of rubber so as to give the impression that hearing on this side has been excluded. If under these conditions the patient fails to hear the spoken voice, as often he feels he ought, then the falsity of his answers is exposed.

Commentary.—As I feel that it would be wrong to give the impression that the subject of the post-concussional syndrome should be approached with a sense of suspicion, it has not been my intention to give a complete account of malingering. Rather I have attempted to indicate an atmosphere than to make an exhaustive study of the problems concerned, particularly as the best results are obtained by conveying to the patient at the outset the impression that you intend to give every side a fair deal and are anxious to make him better. Once antagonism has been aroused by the suggestion of partisanship or by impatience, any therapeutic good that the doctor would otherwise be able to do, either by active treatment or advice, will be nullified. Alternatively, should an obvious inconsistency crop up in the history or clinical examination, then any possible falsity should be probed not only to the patient's but also to the examiner's discomfort

The greatest difficulty in the assessment of the genuineness of symptoms arises in those cases in which the ailments complained of are entirely of a subjective nature, such as headaches. There is, unfortunately, no scientific means of detecting a malingerer under these conditions, one's judgment is dependent purely on the general atmosphere of the case, *i.e.*, on one's clinical acumen. Recently more direct evidence has been forthcoming to prove that abnormalities of electrical potential of the brain can be detected in patients suffering from post-concussional sequels.[1] Possibly the solution of this problem will finally be found in electro-encephalography.

It is, of course, very much easier to understand the later complaints of a patient if the acute phases of his illness have been witnessed, as I have often been impressed by the essential similarity of concussional and post-concussional phenomena. For nearly every feature in the post-concussional period there is a corresponding one in the acute phases of the illness, the difference being purely that of intensity Surely the common sequel of change of temperament must be regarded as the aftermath of the classical stage of irritability.

[1] Williams, D Paper read at the meeting of the Neurosurgical Society, Oxford, July 1941

THE PROBLEMS OF LITIGATION

In cases of litigation the judge in charge of the inquiry will want to know from the doctor :—

1. The nature and severity of the injury complained of.
2. The injured man's present physical and mental state.
3. What the future is likely to show.

As a rule a precise answer can be given to the first question and a satisfactory one to the second. The difficult problem is that of prognosis, and it is with this that the law is chiefly concerned.

Prognosis, of course, is merely another word for prophesy, and in particular the problems to be discussed or settled are :—

1. Will the injured man recover completely, and if so, when ?
2. If he will not fully recover, what will be his disabilities ?
3. When will he be able to start work ?
4. Will he be able to do his own work, and when ?
5. If he is unable to do his own work, what kind of work will he be capable of ?
6. What kind of medical treatment ought he to have, and what expense will this entail ?

The doctor will find himself ill-prepared if he is not ready to answer these questions or at least be able to give a good reason why he is not. He must, of course, never be persuaded to make statements which he cannot substantiate, and must show meticulous care when dealing with facts, otherwise doubt will be thrown on his adductions, and naturally so. The law, it must be understood, is essentially out for the truth and nothing else, and the doctor must always work from this premise

When fortified with all the available data on the nature and severity of the injury and with up-to-date neurological findings, a doctor is justified, but not until then, in giving an opinion on prognosis based on his own experience and on those of other observers [1] who have made a special study of the subject.

First let us consider the type of case in which there is a discrete injury and six months or more have elapsed since the accident.

Loss of Smell.—The resulting disability of loss of smell is obvious, particularly in occupations such as those of chemical workers and cooks In ordinary human beings it adds another hazard to an already sufficiently jeopardised life inasmuch as a

[1] Symonds, C P "The Effects of Head Injury remaining after One Year" Rapport presenté a l'occasion due VII[e] Congrès International des Accidents et des Maladies du Travail Bruxelles, Juillet, 1935

patient cannot detect escapes of gas or recognise unwholesome foods before they enter his mouth. Also, as Leigh[1] has pointed out, anosmia is commonly associated with that form of frontal injury which leads to prolonged ill-health and often to permanent disability.

Loss of Taste.—Complete loss of taste is usually due to irremediable injury of the olfactory pathways. Partial loss may be caused by (i) partial olfactory damage, (ii) contusion of the cauda tympani and great superficial petrosal nerves and (iii) trigeminal denervation of the mouth. The sensations of salt, sour, bitter, sweet, are conveyed by the cauda tympani and great superficial petrosal nerves, and if these are injured the sense of taste is seriously impaired. Taste, as the ordinary person knows it, is a complex stimulus, being dependent for its finer discriminations on the background of common sensation supplied by the trigeminus[2] Therefore in whatever way the nerve supply to the mouth and upper pharynx is affected, the sense of taste is interfered with. Apart from loss associated with facial paralysis, impairments of the sense of taste rarely recover completely. Ageusia destroys one of the primary pleasures of life. Moreover, as patients are constantly reminded of their disability, they often become introspective and miserable.

Diplopia.—Most cases of diplopia clear up within six months of injury, and even after this date the majority recover. In my experience the only cases which remained permanent were those due to deformity of the orbit or to fracture lines cutting across the sphenoidal fissure. Even in doubtful cases it is fair to state that the final prognosis is good

Permanent diplopia is a serious disability, for although double vision can easily be corrected by shading the affected eye, this results in the loss of binocular vision or the three-dimensional view which is essential for the judgments of speed and distance and for many of the finer types of manipulative work.

Aphasia.—Motor aphasia is a very crippling disability not only in itself but in the adverse effect it has on the emotional stability of a patient. Often though he can think quite clearly he becomes obsessed with his difficulty, thinking of nothing else. Sensory aphasia is even more serious as it constitutes a frank intellectual impairment.

Following Closed Injuries—When aphasia is discovered as soon as a patient can co-operate, the underlying pathology is usually a diffuse surface contusion. Given that depressed fractures

[1] Leigh, A D Paper read at the meeting of the Neurosurgical Society, Oxford, July 1941
[2] Rowbotham, G F "Observations on the effects of Trigeminal Denervation" *Brain*, 1939, 62, 364

are raised immediately or clots on the surface of the brain removed early, the prognosis is good. When reviewing these cases it is useful to remember that the ultimate recovery of nervous tissue from an injury takes any time up to two years. Therefore when prognosis is in doubt, a final opinion should be withheld until this time has elapsed

As a generalisation it may be said that following correct surgical procedure, improvement in most cases of aphasia is likely if special training can be carried out afterwards. This usually entails a considerable financial expense which should be considered in the final assessment of damages. When aphasia develops after an interval it is most commonly due to thrombosis and becomes worse rather than better. Theoretically it may be caused by a subdural hæmatoma or cerebral œdema, but these conditions are rare and can easily be confirmed or corrected by suitable measures

Following Open Injuries.—Aphasia resulting from laceration and destruction of the brain tissue is permanent. Delayed aphasia may be due to thrombosis, but we are now seeing cases resulting from secondary abscess following osteomyelitis of the skull. The prognosis in this latter group depends on the success with which the abscess is drained or removed and on how much permanent damage was done before surgical treatment was started.

Defects in the Visual Field.—The resulting disability of a defect in the visual field depends on its extent and whether or not macular vision is affected. When central vision is intact the danger of a blind peripheral area is that injuring objects may approach or be walked into in a way that would not happen in the ordinary course of events In particular, when driving a motor car the extra danger both to the driver and other people is obvious. When central vision is affected the consequences are much more serious to the patient, since reading, writing or close work may become difficult or impossible.

Spastic Paralysis.—The disability of any degree of spastic paralysis of the arm or leg is easy to assess. Often it leads to complete incapacity, and a man has to undergo vocational training for some other and more suitable employment.

Sensory Loss of Cortical Type.—Sensory loss due to injury of the parietal lobe is not only an unpleasant sensation but also leads to locomotor impairments. The fingers are clumsy, finer movements are impossible and the hand becomes virtually paralysed The foot stabs the ground if the movements of the legs are not under the control of the eyes, and walking in the dark becomes dangerous.

In other words, sensory loss of the cortical type in a limb has

the same results to a lesser degree as spastic paralysis. Damages may be given on this basis.

Defects in the Skull.—A defect in the skull of reasonable dimensions associated with an intact dura mater and in the absence of infection is not a serious disability, since it may be repaired by simple surgical means which offer excellent chances of an adequate cure. In wounds, however, that are not already soundly healed or that did not heal by first intention, the outlook is quite different. Reopening of the wound may activate latent infection lurking in the deeper tissues which nullifies any attempt at grafting and which, in fact, may leave the tissues in a worse state than before.

When overlying a dural defect a hole in the skull is always serious, because operative repair involves considerable interference with the surface of the brain. The importance of a simple defect in the skull is obvious. In sedentary and most other forms of life the brain can be protected from danger by an external shield, the patient being able to carry on in the usual way. On the other hand, without operation athletes are precluded from playing the more vigorous forms of games. Whether professional or amateur, the results may be far-reaching either from the financial or psychological point of view Also, a man with a hole in his head finds it much more difficult to get employment than one who is not so afflicted. Superimposed calvarial and dural defects commonly lead to partial but permanent incapacitation.

Symptoms unassociated with Neurological Signs.—The second and larger group is made up of those patients who complain of headaches, dizziness, nervousness, irritability, loss of concentration and insomnia, but who have very little to show for their ailments in the way of physical signs.

This is the most difficult type to assess and the one around which a most heated controversy revolves. In previous sections of this chapter attempts have been made to indicate the underlying pathology of each symptom of the post-concussional syndrome and the conclusion arrived at was that the final state of a patient results from a complicated integration of functional and organic influences, each symptom interacting with the others with adverse effects It has been my experience in the majority of cases that it is useless to start any kind of formal medical or surgical treatment until the problems of litigation have been finally settled. This does not mean, as the representatives of some insurance companies so firmly believe, that their so-called "golden ointment" will cure all ills It will not. The desire for compensation of a man who has received an injury through no fault of his own is a natural one, and the sooner he is relieved of his sense of hurt the

better. Theoretically the assessment of any case would best be made at the end of a man's life, when the effects of the injury could be reviewed in retrospect. In practice I believe that problems of litigation should be settled not later than six months after the accident if there are no special medical reasons why this should not be done. The arguments in favour of this belief are as follows :—

1. Prolonged worries arising from litigation are avoided.
2. A poor man does not have to live on charity with its demoralising effects.
3. A rich man need not immediately lower his standard of living.
4. The patient can procure those extra comforts in life which he so often thinks are necessary for recovery.
5. He can seek special medical treatment.
6. A man is more likely to readjust himself to his new physical and mental state when thrown on to his own resources than when he regards his state of health as being the responsibility of other people.
7. The attitude of the family changes. Whereas the wife previously sympathised with every complaint and condoned any kind of laziness, she now takes a firmer hand and insists on her husband trying to do something useful and resists his tantrums

When discussing the assessment of a case, not only has the nature of the residual illness to be considered but also the man's normal circumstances and the type of work he will be expected to do. It is poor judgment to send a man back to heavy and noisy work amongst machinery if he is suffering from headaches and dizziness Also, it is equally useless to advise a business man to return to a post where responsible decisions have to be made if he is nervous and has lost his confidence A special difficulty arises when a man, rendered unfit for his own employment, is still capable of doing some other kind of work but cannot bear the thought of change and refuses to make the attempt. To compel any one other than a young person to do so will result in failure without prolonged vocational training. In any case, a change in work not the outcome of one's own desire is usually depressing rather than stimulating if there are no financial or other advantages to be gained thereby. Experience has shown that sympathetic and light employment rarely succeeds in getting a man permanently back to work Soon he develops an inferiority complex and feels that his position is unstable, which in fact it is. The good will of employers not directly responsible for compensation is apt to break down. Also, a man in sympathetic employment

is afraid to exert himself unduly, since he may be regraded and lose his partial compensation.

Therefore in view of all the above considerations it is better in the long run to make an early and final settlement even of those cases which come under the Workmen's Compensation Act. To let civil cases run on for a long time in the hope of recovery is usually a mistake, since under these conditions many patients develop further functional complications, growing worse rather than better. In my experience, following severe injuries to the head, it takes about six months after litigation has been settled for a man to readjust himself to his new mental and physical state sufficiently to do useful work, and about three years altogether before he is free from symptoms.

TREATMENT

Given that there is no evidence of circulatory, respiratory or metabolic disease, and that there are no neurological signs to point to a large focus of organic damage in the brain, treatment consists essentially of rehabilitation, followed by readjustment

Rehabilitation.—Rehabilitation consists of graduated physical training, exercises in balance and occupation of leisure hours in useful and interesting pursuits or hobbies. In the Services this is a relatively easy problem, because the men who come up for treatment are of about the same age, conforming to a uniform physical standard. Where civilians are concerned the difficulties are much greater, because a heterogeneous group of patients from a normally energetic boy to a slowly moving, heavily built person of middle or past middle life has to be treated. It is none the less always possible to drill people within the limits of their physical capacities if sufficient workers trained in this type of therapy can be employed or persuaded to give their services free. A housewife may, in the early stages, be required to make the beds in the ward, clean or cook ; a middle-aged man may be given work in the garden. Brisk walks into the fresh air each day under supervision are also important. In cases of dizziness, exercises in balance are an essential form of treatment. Each day a patient must be asked to stoop, rotate and move quickly from one position to another. At first grievous complaints will often be made against the imposition of these tasks, but with encouragement and perseverance these exercises can be prolonged and carried out more frequently with most beneficial results. Careful judgment, of course, must be shown in the choosing of physical tasks, but when this has once been done it is justifiable in the interests of the patient to bring pressure on him to carry them out.

Therapy in leisure hours is just as important as in the more active phases of treatment. It is wrong to allow patients to sit idly around for long periods doing little else than brooding over or comparing their ailments. At least their fingers can be kept busy by woodwork or needlework according to sex. In the right people reading should be encouraged, and the cinema and dancing will be found excellent forms of occasional entertainment. Probably criticisms will be levelled at the rehabilitation schemes that are now being developed by the Emergency Medical Service as being over-elaborate, expensive, and that they cultivate in a patient a taste for hydro life. I believe that time and results will prove these views to be incorrect. One essential factor in the success of a rehabilitation centre is that the man in charge of the clinic works with conviction and supervises his cases personally.

Readjustment.—At the end of, say, a month a review should be made of progress and a decision made whether a further course of treatment is necessary or whether the patient is fit to return home and to work. When he is discharged he should not be lost sight of. Ideally, each patient should be followed into his home by the representative of some society interested in this work to see that, as far as is possible, the right conditions are obtained. Obviously it is wrong to send a relatively ill woman home to take care of a large family of young children without some kind of help. A patient, on the other hand, must not be taught to regard herself as an invalid, and the people around her must be told not to treat her as such. In the case of a working man, efforts must be made to find him the right kind of employment and to give him the assurance that attempts on his part to work will not necessarily result in his being written off as completely fit at the whim of some irresponsible person. In civil cases, when a patient has received a large sum of money in compensation for his injury, it would often be better if the money was kept in court, being dispensed as a responsible body thought fit. In the case of children, the money should be spent in bringing them up to the standard of education from which they have fallen, as a result of accident. Older people often realise too late, when most of their money has disappeared, that the success of so-called "small" businesses, such as newsagents and sweet shops, entail long hours of work and diligence

Vocational therapy means the training of a man for some kind of employment within the capacity of his physical disabilities when he has been rendered unfit by accident to do his own work Such training is rarely necessary and has only to be carried out in the type of injury which results in a frank physical defect

Medical Therapy.—Meals should be taken at regular hours and in quantities that are easily digested, but few restrictions need be put on the type of food taken, the choice depending largely on the patient's personal tastes. Smoking is best curtailed to a half-hour period following meals and definitely forbidden on an empty stomach as this is, in itself, apt to cause giddiness. Save in the habitual spirit drinker, and here rigid restrictions should be made, alcohol in any form should be forbidden. The vaso-dilatation which results from imbibing alcohol often precipitates symptoms. Also, the effect of strong liquor is so heightened that a patient may lose control of his judgment when taking even smaller amounts than those which left him unaffected in his pre-accident days. The non-realisation of this may lead a patient into serious trouble when in charge of a motor car.

A bad sleeper often develops the habit of going to bed late in the hope that sleep will come to him earlier. This belief leads to a vicious circle of irregularity and to the demoralising effect of rising late in the morning. Early to bed and early to rise is a strict injunction that must be given to every patient suffering from post-concussional insomnia. Drugs to procure sleep are permissible for short periods only and should be withdrawn after a few weeks as an addiction to barbitone and paraldehyde is very soon developed. Bromides to damp down nervous tendencies, in my experience, do more harm than good because of their depressing effects. On the whole it is better to give tonics, such as neurophosphates, rather than medicines to calm the nerves. Aspirin and Veganin should be given only when pains are severe and the good effects of the drug obvious. When headaches are of the migrainous type, ergotamine tartrate may be necessary to give relief. Following compound wounds, luminal ($\frac{1}{2}$ gr.) should be given every night for six months as a prophylactic measure against epilepsy.

The essential object of medical therapy is to see that a patient lives as normal and healthy a life as possible and to effect improvement by measures chiefly directed to the general health. In cases of asthenia, sunlight and hydrotherapy will often be found of great value. Also, vitamins should be given in generous quantities. To neglect medical therapy because the measures are simple is a very grave error in judgment and one that throws patients into the hands of nature curers.

Psychiatrical Therapy.—Much can be done for a neurotic patient without the aid of formal psychiatrical methods if his doctor is interested and willing to spend time in helping him to solve his problems. Occasionally an injury merely precipitates a mental state, the result of previous dissatisfaction, of which

the doctor may become aware in ordinary conversation if the patient is given time to come out with it. Recently a woman came to me complaining of weakness and pains in her arm following a motor car accident in which she was only slightly bruised. All kinds of active treatment failed to do her any good. On talking to her during treatment it was found that her trouble was due to domestic and family worries rather than to post-concussional influences. Not until these conditions have been corrected is improvement likely.

When a background of neurosis previous to accident is suspected, but the reason for which is vague, then a patient should immediately be passed over to a psychiatrist. No doubt as the need becomes more widely recognised, psychological overhauls will become a routine in the investigation of the post-concussional syndrome in peace as well as in war time.

Surgical Treatment—*Lumbar Puncture.*—Before embarking on treatment of post-concussional symptoms the pressure of the cerebrospinal fluid must be measured by spinal manometry, since it is axiomatic that intracranial tension must lie within normal limits if the optimum conditions for the recovery of cerebral tissues are to be established. On those rare occasions when cerebrospinal fluid pressure is high, and there is no obvious intracranial lesion to account for it, spinal drainage or intravenous dehydration associated with restriction of intake of fluids is necessary When fluid pressure is low and leakage through the nose or ears cannot account for it, fluids in excess of the normal requirements of the body should be taken by mouth. Also, care should be taken to see that adequate amounts and the correct proportions of inorganic salts, such as sodium chloride, are included in the diet. When symptoms are persistent, repeated manometric tests ought to be made each week.

Encephalography—The indications for encephalography in the post-concussional syndrome are as follows :—

1. When there are signs of local brain damage, such as spastic paralysis, etc.
2. In cases of persistently raised intracranial pressure
3. When, in spite of treatment, a patient does not improve.
4. In cases of frank mental deterioration.
5. When the development of air encephalopathy is suspected.
6. For the treatment of adhesions causing traction headaches in an endeavour to break down the adhesions

Local Explorations.—In cases of persistent and localised pain or the development of secondary headache, a trephine hole should be placed over the painful area and the subdural space explored.

By means of a flat metal retractor passed along the space, collections of fluid may be tapped or troublesome adhesions broken down. This is a form of treatment which is far too often neglected, and if the trephine disc is replaced at the end of the proceeding the patient is not left with a defect in his skull.

Subtemporal Decompression.—A subtemporal decompression is rarely needed. In my series it had to be done only in those cases when intracranial pressure remained abnormally high in spite of spinal drainage or dehydration and when the underlying pathological state could not be diagnosed or treated directly. If done indiscriminately as a last measure it will only lead to further complaints rather than to the relief of symptoms. Also, it leaves a defect in the skull which the patient can use as a lever for the extraction of compensation.

Defects in the Skull.—When associated with symptoms either of organic or psychological origin a simple defect in the skull should be repaired without hesitation

Eighth-nerve Section.—Eighth-nerve section in the posterior fossa is an operation of considerable magnitude, being necessary only on the rarest occasions. Its indications are frequent attacks of severe vertigo of proved labyrinthine origin which cannot be controlled by conservative measures.

Plastic Operations.—It is wrong to belittle a deformity which is obviously worrying a patient. Crooked noses should be straightened, deflected septa resected and ugly scars excised and repaired

TRAUMATIC OSTEOMYELITIS

The bones of the vault of the skull are composed of two tables of compact osseous tissue enclosing a diploe. Diploe is merely another name for cancellous bone The only way in which this differs from the medulla of other bones is the presence of large venous channels which course through it These, as far as I have been able to discover, have no lining membrane of soft tissue. Also, they are concerned rather more with the circulation of the brain and scalp than with the metabolism of the bone itself. They drain (i) into the dural venous sinuses, (ii) into the meningeal veins and (iii) into the veins of the scalp It will be seen, therefore, that there are wide channels of access for possible infection directly from the scalp to the brain. This is the reason why even simple wounds of the scalp should be treated carefully.

The outer surface of the bone is covered by pericranium. This membrane separates easily, save at the suture lines where it is firmly attached The endosteum or inner lining consists of the outer layer of the dura mater. This also strips readily, save at the

margins of the basal foramina and at the points of entrance of nutrient vessels.

The infective complications which follow injuries of the paranasal air sinuses and petromastoid bones have been discussed in Chapters V and VI.

Osteomyelitis of the vault,[1,2] which is a much less serious complication than the above, is the subject with which we are concerned here.

Acute fulminating osteomyelitis associated with toxæmia, septicæmia or pyæmia, such as arises in the juxta-epiphyseal region of the long bones of young boys, is almost unknown in the vault of the skull. Occasionally a severe osteomyelitis of the frontal bone may result from an acute suppurative frontal sinusitis, but this rarely follows injury.

OSTEOMYELITIS FOLLOWING CLOSED INJURIES

Osteomyelitis of the vault of the skull following injuries in which the scalp remains intact is rare. When it does occur it is always of the chronic or subacute variety. The sequence of events is as follows: a hæmorrhage occurs between the outer table and its covering membrane, with the resulting formation of a localised subpericranial clot. This becomes infected either from a damaged hair follicle or through the blood stream. An abscess occurs, with the result that the superficial layers of the outer table become infected. Clinically this process declares itself as a boggy swelling beneath the scalp to form a typical Pott's puffy tumour. If left undisturbed the abscess enlarges, bursts through the skin and a fistula results.

Treatment in these cases consists in opening the wound widely, drainage of the abscess and removal of any loose flakes of bone. The wound is then packed with powdered sulphanilamide or sulphapyridine and allowed to granulate from its base. Measures to improve the general health should also be instituted, since a Pott's puffy tumour is evidence of poor tissue resistance to low-grade infection.

If healing does not rapidly take place the following conditions should be suspected (i) tubercular osteitis, (ii) syphilitic osteitis, (iii) metastatic tumours and (iv) hæmapoietic diseases.

Aseptic Necrosis.—As the name implies, aseptic necrosis is not an inflammatory process. It is due to the destruction of an epiphysis by trauma and occurs in children, leading to large defects in the skull

[1] Wilensky, A O "Osteomyelitis of the Skull" *Arch Surg*, 1933, **27**, 83

[2] Adelstein, L J, and Courville, C B "Traumatic Osteomyelitis of the Cranial Vault with Particular Reference to Pathogenesis and Treatment" *Arch Surg*, 1933, **26**, 539.

OSTEOMYELITIS FOLLOWING OPEN INJURIES

Direct contamination of an open wound is the usual cause of traumatic osteomyelitis. Its occurrence depends almost entirely on the efficiency with which compound fractures of the skull are treated in the early stages. With adequate facilities and early treatment, primary healing in air raid casualties and civil injuries is somewhere about 100 per cent.

Localised Osteomyelitis.—There are three clinical types in this group :—

1. A wound that has apparently healed becomes sensitive to pressure. Then a swelling appears, a length of the scar opens and a discharge of pus occurs. There are no signs of toxæmia, but the wound in spite of careful dressings continues to discharge.

Radiography, though often negative, may show the presence of a foreign body or of a superficial erosion of the outer table.

In these cases the wound should be widely opened if it does not heal rapidly. Commonly, foreign bodies, such as dirt, grit, hair or pieces of clothing, will be found in the deeper tissues. These are removed and granulation tissue is cut away. If the surface of the bone is roughened it should be scraped and small sequestra removed. The wound is then packed with sulphonamide powder as described above.

2. Sometimes as a result of infection a wound is not consolidated by primary healing. When infection is thought to arise in the deeper tissues, as judged by a copious amount of discharge and surrounding œdema, then the wound should be opened and treated as in (1) above. To wait for any length of time in the hope that the wound will heal without further intervention is usually a waste of time. Simple infection of the edges of a skin wound shows signs of improvement within a few days.

3. Occasionally the edges of a defect in the skull become infected, resulting in a sclerosing osteitis and a persistent discharge of pus through one or more fistulæ. In these cases radiography shows that the edges of the bone are irregularly sclerosed. At times discrete sequestra are demonstrated.

Treatment consists either in block resection of the infected bone or removal by piecemeal nibbling. In most of these cases it will be found that conservative measures only are necessary to clear up the discharge. When operating, great care must be taken not to pierce the dura whichever method is used, as meningitis may be the result To avoid opening the meningeal spaces the dura should be separated carefully from the bone by means of a curved dissector The amount of bone to be nibbled away can be judged by the extent of the dural granulation tissue, which

must be completely uncovered in all directions. Unhealthy granulation tissue is removed by gentle sweeps with a gauze swab.

In some cases a dimple or thickening of the dura will suggest the presence of an intradural abscess, which was not suspected on clinical grounds. By gentle finger pressure on the surface of the brain a bead of pus may be made to well up through a fine fistula. When this happens the opening should be enlarged and the underlying abscess drained. In cases of doubt, intradural exploration should not be done until the presence of an abscess has been demonstrated by encephalography.

Infection of Loose Fragments.—In the débridement of large compound wounds it is sometimes justifiable to leave loose fragments of bone in position so as to minimise the resulting defect. If at any time when this has been done infection occurs, the wound should be reopened immediately and the loose fragments removed. After packing of the wound with sulphonamide, large skin flaps may be loosely resutured if an adequate opening, say 1 in. or so, is left for drainage.

Spreading Osteomyelitis of Subacute or Chronic Type.—An indolent osteomyelitis may, as the result of thrombophlebitic processes, travel extensively along the diploic spaces. At one point the outer table may necrose; here an external fistula may form At another point the inner table may sequestrate, here an extradural abscess may form. Finally the whole segment of the affected bone takes on the classical worm-eaten appearance, the X-ray showing a fluffy area of irregular rarefaction enclosing dense sequestra.

Treatment is difficult because :—

(i) Large areas of bone are affected.
(ii) It is impossible to know by clinical or radiographic means just how far the inflammatory processes extend.
(iii) The overlying skin is unhealthy; it is riddled by multiple sinuses and in places may be adherent to the bone
(iv) The bacteria concerned are tenacious; or the tissues have a predisposition or lack of resistance to the organism concerned Such conditions are very similar to tubercular infections of bone in their persistency.

Theoretically, the best treatment is complete removal of the affected segment by block resection through healthy bone. The objections to this method are :—

(i) A large defect in the skull would be the result
(ii) The danger that a large skin flap necessary for the exposure would not heal.

(iii) The difficulty in knowing how much bone to resect in order to cut beyond the boundaries of the infection.

(iv) The danger that infection may start in the edge of the cut bone, however wide a resection be made.

In extensive infections it is probably wiser to approach the affected bone by means of a series of vertical parallel cuts through the scalp and to undermine the rectangles of skin so formed. In this way an unlimited area of the skull can be exposed without the danger of the skin retracting in the same way as when a flap fails to heal. The whole of the external table is then removed over infected diploe; granulation tissue is scraped away and necrosed areas of the inner table excised. The wound is then packed with sulphapyridine; later a course of sunlight or deep X-ray therapy is applied to the head. Medical measures to bolster up the general health are also important. In fact this is the type of infection which often necessitates treatment in a sanatorium.

COMPLICATIONS

These are: (i) extradural abscess, (ii) meningitis, (iii) cerebral thrombophlebitis, (iv) encephalitis and (v) cerebral abscess.

Any of these conditions may result from a severe compound fracture or what appears to be a simple wound of the scalp. In the latter case the typical sequence of events is as follows A child receives a simple blow on the head, say with a piece of slate, which lacerates the scalp. This is stitched or dressed and the child allowed to go home. Within twenty-four hours he complains of headaches and soon is seriously ill with some kind of intracerebral complication.

Early treatment is essential if the child is not to die. Chemotherapy is, of course, started at once by mouth, but surgical intervention is also necessary. The wound should be reopened and the dura exposed by removing a piece of bone with a large trephine. If from the appearance of the dura, *i e*, by thickening, granulation tissue or a fistula, the route of infection can be determined, the dura should be opened when pus may be released. If a negative exploration results, no harm will be done if the wound is packed with sulphapyridine.

The treatment of cerebral abscess depends on whether or not the abscess is attached to the dura When attached to the dura the abscess should be opened and drained. When subcortical in position, it should be aspirated as often as is necessary, then excised at a later date.

THE FINAL RESULTS OF HEAD INJURIES

The majority of injuries to the head are in the nature of simple scalp wounds or abrasions. These rarely lead either to complications or sequels.

Occasionally a simple blow may produce an apprehension neurosis or precipitate epilepsy or cause a subdural hæmatoma.

Following gunshot wounds of the head the high incidence of epilepsy is extremely disturbing, most writers on this subject placing the figure between 30 and 40 per cent. Also, many of the afflicted die at an early age.

In air-raid casualties a large number of simple scalp lacerations and abrasions are occurring. These probably will not give rise to complications Large numbers of compound depressed fractures of the skull without penetration of the dura are also being treated. As the majority of these heal by first intention and are unassociated with obvious damage to the brain, I believe that sequels will be minimal. A final opinion on this point, however, will have to be withheld for at least another three years. The outlook is much more serious in compound wounds of the brain and the figures of the last war may help in an assessment of these cases. Patients from past raids who have been operated on are now coming up to Out-Patient Departments for review. The spirit of these people is outstandingly good. Few are unduly nervous Most are anxious to do more than they did before they were injured. Whilst undergoing treatment, even those with multiple injuries are anxious to get out of hospital to look after their families This anxiety to return to duty is not so obvious in Service patients. These, like litigants, are apt to nurse their ills. The patients who do worst of all are those who know that pieces of metal are embedded in their brain.

Compound fractures due to road or industrial accidents are relatively infrequent. Closed head injuries, on the other hand, are exceedingly common. Of these, 16 to 25 per cent. die of their injuries Those who recover are incapacitated from periods of three months to three years At least two in every hundred never work again or live a life of semi-invalidism. The incidence of epilepsy lies between 0 1 and 2 5 per cent.

The final results of trauma to the head can only be appreciated if the injured are followed into their homes at long periods after accident It will then be found that actual physical disabilities, such as paralysis, are rare None the less the power to work at the pre-accident standard, or indeed at all, may have been destroyed An interesting discovery I made whilst interviewing many of the cases of my epileptic series was that although people with no

compensation at stake started work earlier than those who were entitled to compensation, the non-litigants more frequently broke down later to become totally incapacitated.

It will have been realised by this time that the immediate and late results of acute cerebral trauma are serious not only for the injured but also for the State With correct surgical and medical procedure in the first instance, many more lives could be saved and sequels minimised by adequate rehabilitation. Economically much expense could be saved for the country if facilities for readjustment were elaborated

From the point of view of prevention, much could be gained by improved legislation. One of the main causes of dangerous driving, *i e.*, driving dangerous to other people, is the knowledge of the driver that he is relatively safe Few drivers are killed Moreover, a driver may pass his financial liabilities resulting from an accident over to an insurance company for a small annual sum of money. An excellent deterrent to thoughtlessness would be to force him to cover compensation to some extent from his own wages —say 25 per cent. of these—until the bill is paid. The dangers of injuries to the head in motor cycling may be minimised by the compulsory wearing of crash helmets.[1]

These various suggestions may appear mundane, but philosophy can play very little part in therapeusis. At best it is a statement of conscious ignorance, interesting to the reader and flattering to the writer. Probably time, and sooner rather than later, may alter this view.

[1] Cairns, H "Head Injuries in Motor Cyclists" *Brit Med Jour*, 4th October 1941, 465

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